JAN 7 1959

UNIVERSITY OF MICHIGAN
DENTISTRY LIBRARY

Volume 14 No. 1
January • 1959

c.2

THE AMERICAN ACADEMY OF DENTAL MEDICINE

COOPERATION KNOWLEDGE UNDERSTANDING

FOUNDED 1945

ARTICULOS SUMMARISATE IN INTERLINGUA

journal of dental medicine

OBJECTIVES OF THE AMERICAN ACADEMY OF DENTAL MEDICINE

(Quoted from the Constitution)

"To promote the study and dissemination of knowledge of the cause, prevention and control of diseases of the teeth, their supporting structures and adnexa and related subjects; and to foster and promote a better scientific understanding between the fields of dentistry and medicine."

IN THIS ISSUE

THE NATURE AND CAUSATION OF HEADACHE. Harold G. Wolff 3

THE DENTIST AND VOCATIONAL REHABILITATION. Howard A. Rusk and Samuel D. Herman 35

CONGENITAL DENTAL DEFECTS. Seymour J. Kreshover 41

SURGICAL PROCEDURES IN PERIODONTAL THERAPY. Harry Roth and Howard L. Ward 49

CASE REPORT: AMELOBLASTOMA OF THE MANDIBLE. Harry Fredrics. David L. Hemphill, and Irwin W. Scopp 54

BOOK REVIEWS 58

ACADEMY PROCEEDINGS 60

ANNOUNCEMENTS 64

JOURNAL OF DENTAL MEDICINE

VOL. 14 JANUARY, 1959 NO. 1

Editor
Irving Yudkoff, 57 West 57th Street, New York 19, N.Y.

Consultant Editor
Allan N. Arvins

Associate Editors
J. Lewis Blass
Marvin N. Okun
S. Leonard Rosenthal

Assistant Editors
Sanford C. Frumker
Henry I. Greene
Alexander Soberman
Nathan Wachtel
George J. Whinston
George J. Witkin
Arthur N. Yohai

Business Manager
Harold E. Brennan, 400 Madison Avenue, New York 17, N. Y.

Publications Committee
Irving Yudkoff, B.A., D.D.S., *Chairman*
Allan N. Arvins, D.D.S.
Morton S. Biskind, M.D.
J. Lewis Blass, Ph.G., D.D.S.
Louis R. Burman, Ph.G., B.S., A.M., D.D.S.
Julius N. Obin, B.A., D.D.S.
Hayes Martin, M.D.
Harry Roth, B.S., D.D.S.
Sidney Sorrin, D.D.S.

* * *

The Journal of Dental Medicine is the official publication of the American Academy of Dental Medicine. It is published quarterly, in January, April, July, and October at New York, N. Y. Publication in the Journal of Dental Medicine is not an endorsement by the American Academy of Dental Medicine of opinions in any article, abstract or extract. The Editor and Publisher are not responsible for opinions expressed by authors of contributions.

Subscription rate: $6.00 Annually, $6.50 Foreign. Single copies, $1.50. Special Nutrition Issue (Sept. 1953) $1.00 per copy, special rate in quantity. Tenth Anniversary Issue (April 1956) $1.00 per copy, $5.00 for bound volume. Special student subscription rate, $4.00 annually.

Copyright 1959 by the American Academy of Dental Medicine, Inc.

Preparations of Scientific Manuscripts

1. Manuscripts should be typed, double spaced, on one side of 8½ by 11 inch bond paper, allowing one inch margins.

2. The title should appear at the top of the first page, followed by the name and degrees of the author or authors. The address of the author should appear at the end of the article.

3. The name of the group before which the paper was presented, the date and place of delivery and the author's university, hospital or government affiliation, if any, should appear at the bottom of the title page.

4. Manuscripts presented for publication should: a—present new facts, describe new techniques or methods of practice or reevaluate established principles; b—contain the results of original research, or c—provide a review of facts concerning a particular subject so that the reader may deduce therefrom legitimate, important conclusions.

5. Data presented in charts and tables should be discussed but not repeated in the text.

6. A concise summary and a precise conclusion add to the value of a scientific article. The summary should state briefly the purpose of the article and the main facts presented or reviewed.

7. Bibliographic references should be in numerical sequence and correspond to the numbers placed in the text where they are mentioned.

8. An article that has been published previously under the same or a different title, or one that contains extensive excerpts from a previously published paper or book, should not be submitted for publication.

Foreign Correspondents

Argentina: Dr. Roberto Lado, Buenos Aires.
Australia: Dr. H. Rich, Sidney.
Burma: Dr. Wu Moon-Aung, Rangoon.
Brazil: Dr. Jacob Reifman, Rio de Janeiro.
Canada: Dr. Mooney Averback, Winnipeg.
Dr. Bruce Squires, Ottawa.
Colombia: Dr. Carlos de Castro, Bogota.
Cuba: Dr. Claudio Funcia Cornell, Havana.
Dominican Republic: Dr. Fernando Camino Cestero, Trujillo City.
Great Britian: Dr. A. Bryan Wade, London.
Greece: Dr. Or. A. Louridis, Athens.
Haiti: Dr. Gerald Roumain, Port-au-Prince.
India: Dr. Om Gupta, New Delhi.
Italy: Dr. Guiseppe Ceria, Turin.
Mexico: Dr. Fermin Reygadas, Mexico City.
Peru: Dr. Rolando Bernui, La Oroya.
Puerto Rico: Dr. Carlos Sosa, Santurce.
Spain: Dr. I. Saenz de la Calzada, Madrid.
Venezuela: Dr. L. J. Mota Potentini, Caracas.

DIRECTORY OF OFFICERS

President	D. Chalmers Lyons, 420 West Michigan Avenue, Jackson, Mich.
President-Elect	George G. Stewart, 1410 Medical Arts Building, Philadelphia, Pa.
Vice-President	Hermann Becks, 2240 Channing Way, Berkeley 4, Calif.
Secretary	Walter I. Levine, 809 Cathedral Street, Baltimore, Maryland
Treasurer	William M. Greenhut, 124 East 84th Street, New York 28, N. Y.
Editor	Irving Yudkoff, 57 West 57th Street, New York 19, N. Y.

CHAIRMEN OF COMMITTEES

*Constitution	Harold R. Gelhaar, Upper Montclair, N. J.
*Dento-Medical Relations	Morton De Scherer, Englewood, N. J.
*Fellowship & Honorary Membership	George G. Stewart, Philadelphia, Pa.
*Membership	Abraham Goldstein, Brooklyn, N. Y.
Planning and Finance	M. A. Archambault, Montreal, Que.
*Publications	Irving Yudkoff, New York, N. Y.
Residency in Dental Medicine	Milton Hyman, New York, N. Y.
Sections	Samuel Turkenkopf, Newark, N. J.
*Historian	William M. Greenhut, New York, N. Y.
*Graduate Awards	Lawrence Staples, Boston, Mass.
Representative, Dental Information Bureau	Anthony Posteraro, New York, N. Y.
Alternate, Dental Information Bureau	Melvin J. Pomeranz, New York, N. Y.
*Program & Clinic—General Chairman	George G. Stewart, Philadelphia, Pa.
Chairman for 1959 Annual Meeting	George F. Clarke, Boston, Mass.
Local Chairman, 1959 Annual Meeting	Rueben Feltman, Passaic, N. J.
Chairman for Mid-Winter Meeting 1958	Nathan Wachtel, New York, N. Y.
Members Outside North America	Claudio Funcia Cornell, Havana, Cuba
Education	Harold E. Brennan, New York, N. Y.
Membership Retention	Joe Barkley, Detroit, Mich.
Ethics	James Ladd, Pontiac, Mich.
Keys	Louis R. Burman, New York, N. Y.
Insurance	William M. Greenhut, New York, N. Y.
Samuel Charles Miller Memorial Fund	J. Lewis Blass, New York, N. Y.
International Interchange of Dental Medicine	S. Leonard Rosenthal, Philadelphia, Pa.
Parliamentarium	Joe Barkley, Detroit, Mich.
Advertising	Horton Kimball, Detroit, Mich.

DELEGATES FROM SECTIONS

Connecticut	Lester B. Weiss, New Haven, Conn.
Maryland	Irving Abramson, Baltimore, Md.
Massachusetts	Earl Stone, Boston, Mass.
Montreal	Lou Rosen, Montreal, Canada.
New Jersey	Abram J. Chasens, Plainfield, N. J.
New York	Sanford Kirsch, White Plains, N. Y.
Philadelphia	Jacoby T. Rothner, Philadelphia, Pa.
Michigan	Edgar James, Detroit, Mich.
Ohio	Samuel Robbins, Cleveland, Ohio.
Texas	Edward T. Farris, Austin, Texas.
North Carolina	J. B. Friedland, Charlotte, N. C.

PAST PRESIDENTS

‡Samuel Charles Miller, New York, N. Y.
Sidney Sorrin, New York, N. Y.
J. Lewis Blass, New York, N. Y.
George A. Bruns, Boston, Mass.
Louis R. Burman, Bronx, N. Y.
‡Gordon R. Winter, Philadelphia, Pa.
Harold R. Gelhaar, Upper Montclair, N. J.
‡Alvin H. Berman, Baltimore, Md.
George F. Clarke, Boston, Mass.
S. Leonard Rosenthal, Philadelphia, Pa.
William M. Greenhut, New York, N. Y.
Marcel B. Archambault, Montreal, Canada.

Section 31 of the Constitution: "The Board of Trustees shall consist of the elected officers of the Academy; the past presidents; the chairmen of the standing committees; and one delegate from each Section." (The President-Elect shall act as Chairman of the Board of Trustees.)

* In accordance with the Constitution.
‡ Deceased.

THE NATURE AND CAUSATION OF HEADACHE*

HAROLD G. WOLFF, M.D.†

Mr. Chairman, Ladies and Gentlemen. . . . It is my privilege to talk to you about headache, a topic of my special interest, and furthermore, the commonest complaint that doctors are called upon to manage. More than 85 per cent of the population in this country have headache at one time or another. Less than 15 per cent of the people say they never have headache at all, and I suspect that some of this group really do have occasional headaches.

Headache always means something wrong, and fortunately for us, it more often means "wrong pace" or "wrong direction" rather than an ominous signal of disease; yet, the quality and the intensity of the experience may be temporarily incapacitating.

What I have to tell you first is about pain sensitive structures in the head. This information has been gleaned from conscious human beings, during operations for serious intracranial disorders and from others who were suffering from headaches and who were able and willing to cooperate in bedside and laboratory observations and describe their experiences. I always embrace such an opportunity as this to express again my thanks for their contributions.

I am going to start by pointing out to you what the pain sensitive structures about the head are, and how they may become the source of pain. I will indicate the numerical frequency and importance of the various types of headache, and finally I will suggest a therapeutic orientation.

In Figure 1 you see the brain, with the skull cap removed. The bone itself is insensitive, although the coverings of the scalp are strikingly sensitive. Wherever you see a solid circle, it means that that part, when stimulated, may cause the subject to complain of pain. The empty circle means the part stimulated was not painful. The cross-hatching on the small outlines of heads at the side indicate the part of the head in which the pain was experienced.

With stimulation along the margins of the superior sagittal sinus anywhere from one end to the other, the subject experiences pain in and behind the eye. Stimulation of the middle meningeal artery and the tributary veins close to the superior sagittal sinus also give rise to pain. The vessels overlying the cerebral hemispheres and the dura are insensitive, with the exception of the areas marked.

Figure 2 shows the cerebellum, the overlying dura and the bone. Again, the bone, the dura and the pial vessels are insensitive. However, the great venous structures, the transverse sinus and occipital sinus are pain sensitive.

The middle meningeal artery, Figure 3, is pain sensitive over its entire course, and when stimulated with electrical current (as was done in all of these experiments) the subject experienced pain over that portion of the head supplied by the first division of the fifth cranial nerve. The brain itself can be torn, pulled, burned, cut, and otherwise injured, without producing pain. (Figure 4).

The large venous and arterial structures at the base of the brain are pain sensitive. These include the circle of Willis from which the major blood supply to the brain originates. On stimulation of these structures, pain is experienced in, behind and over the ear, homolaterally. A few centimeters from the circle of Willis, these great arterial structures become insensitive. (Figure 5).

In general, stimulation of the structures above the tentorium which are supplied by the first division of the fifth cranial nerve results in pain in various regions in front of a line drawn vertically from the ears across the top of the head, whereas, stimulation of pain sensitive

* Presented before the Eleventh Annual Meeting of the American Academy of Dental Medicine, May 31, 1957, Boston, Mass.

† Professor of Medicine (Neurology), Cornell University Medical School, New York, N. Y.

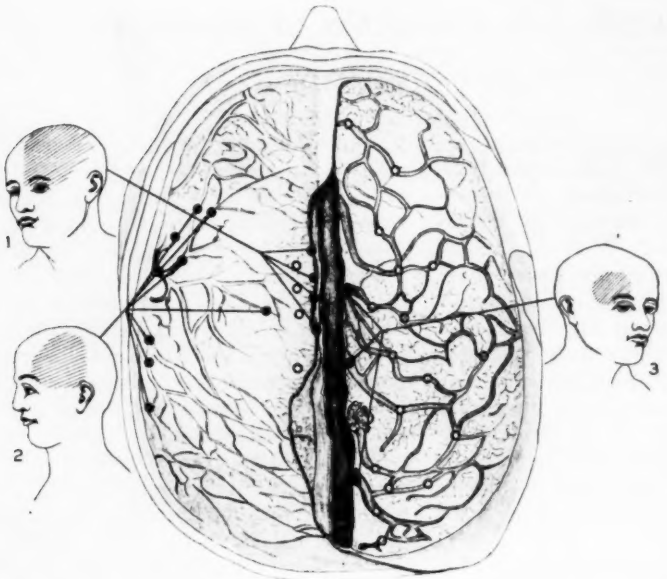

Fig. 1. The calvarium removed. The dura and the meningeal arteries are shown on the left; the cerebral and tributary veins are shown on the right. O indicates the point of stimulation without pain. ● indicates the point of stimulation causing pain to be experienced in the cross hatched areas indicated on heads 1, 2 and 3.

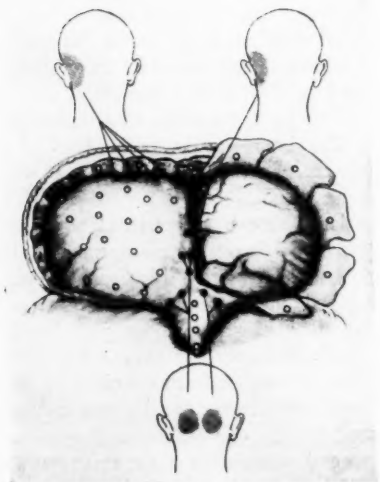

Fig. 2. The venous sinuses and the dura over the cerebellum and the upper part of the cord. O indicates the point of stimulation without pain. ● indicates the point of stimulation causing pain.

intracranial structures on or below the inferior surface of the tentorium cerebelli results in pain in various regions behind the line just described. The latter pain pathways are chiefly in the 9th and 10th cranial nerves and the upper three cervical nerves. (Figures 5 and 6).

With electrical stimulation of the lining and the ventricles of the brain no pain was experienced as indicated in Figure 7. However, when a balloon was introduced into the lateral ventricle in one patient and again into the third ventricle of another patient, and then these balloons were distended, the subjects complained of pain, in the cross-hatched areas indicated in Figures 8 and 9.

We were also able to show that a sudden collapse of the balloon caused pain. Therefore, we came to the conclusion that the painful stimulus was the traction and displacement of the pain-sensitive tributary veins, indicated by the arrows, suddenly pulled upon or displaced by inflation or deflation of the balloon.

In epitomizing what has been said, you have been given evidence for five basic mechanisms

Fig. 3. Middle meningeal artery. O indicates the point of stimulation of the dura without pain. ● indicates the point of stimulation causing pain.

Fig. 4. Arteries over the cerebral cortex. O indicates the point of stimulation without pain.

that operate within the cranium to produce pain:

1. Traction and displacement of the longitudinal and great venous sinuses and their tributaries.

2. Traction and displacement of the middle meningeal artery.

3. Traction and displacement of the great arteries constituting the Circle of Willis.

4. Direct pressure upon major afferent nerves: the fifth, ninth and tenth cranial and the upper two cervical nerves.

5. Inflammation of or about any of these structures that I have mentioned, as well as portions of the pia and dura at the base.

With this kind of information, to what extent can headaches be explained? One erroneous explanation is commonly thought to be related to increase or decrease in intracranial pressure. First, consider certain facts about pressure within the head and pain. If you were to put a needle into the subarachnoid space and raise the intracranial pressure by introducing sterile saline, it would be possible to show that this kind of increase in intracranial pressure to the levels of pressure usually associated with brain tumors, does not cause headache. It may cause a number of disagreeable sensations in the back and legs, but not headache.

This is in keeping with the facts in Table I which point out that in subjects with brain tumor headache there is little relationship between headache and elevated intracranial pressure. Headache occurs almost as commonly in those patients with normal intracranial pressure and brain tumor as it does in the patients with increased intracranial pressure and brain tumor. Out of 72 subjects, 65 had headache. The pressure was normal in 23, and headache occurred in 19 or 83 per cent, whereas the pressure was increased in 49, of whom 46 had headache, or 94 per cent. Thus, almost as large a percentage

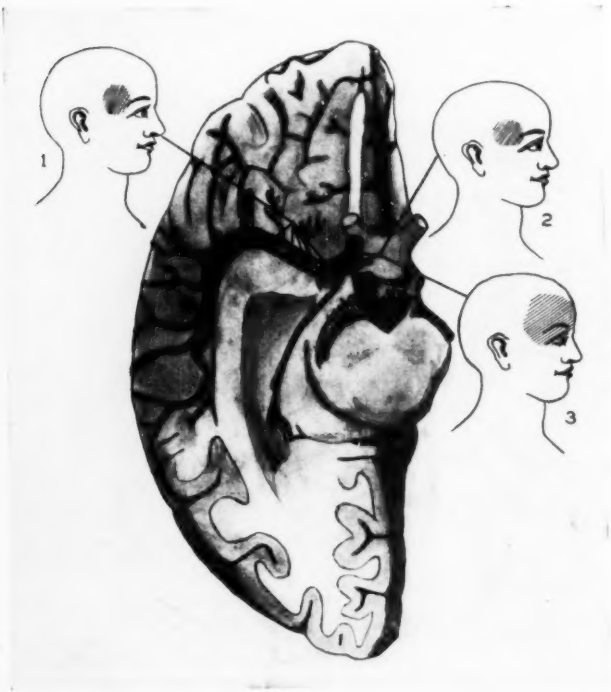

Fig. 5. Intracranial portion of the internal carotid artery and the proximal part of the middle cerebral artery. O indicates the point of stimulation without pain. ● indicates the point of stimulation causing pain.

TABLE 1—BRAIN TUMOR AND INTRACRANIAL PRESSURE

Headache occurs almost as commonly in those patients with *Normal* intracranial pressure as it does in those paitnts with *Increased* intracranial pressure.

Total Cases	*72 Headache*	*65 Headache*	*No 7*
Normal Pressure	23	19	4
Increased Pressure	49	46	3

of patients with normal pressure had headache as did those with increased pressure; therefore, headache could not be attributed to pressure *per se*.

What then, are the relevant facts? In Figure 10, I will attempt to point out what I think is important. In the upper left of this figure you see epitomized the story of a man with headache of approximately two months duration. He experienced headache in the cross-hatched area. At operation, it was revealed that this man had a meningioma, which directly overlay the groove of the middle meningeal artery. By manipulating this tumor, it was possible to reproduce the headache shown in the cross-hatched area and we concluded that the headache resulted from traction on and displacement of the middle meningeal artery.

Now, consider the lower right hand corner of Figure 10. This patient, with a brain tumor in the posterior fossa, experienced pain behind the ear. It was due to traction and displacement of a number of structures, the internal auditory artery, the inferior surface of the transverse sinus, the pontile arteries and the dura.

In other words, in these patients with brain tumor and normal intracranial pressure what caused their pain was traction and displacement of pain sensitive structures, since pressure increase could not be implicated.

Figure 11 carries the argument a step further. A little device is pictured which was developed for the purpose of defining the com-

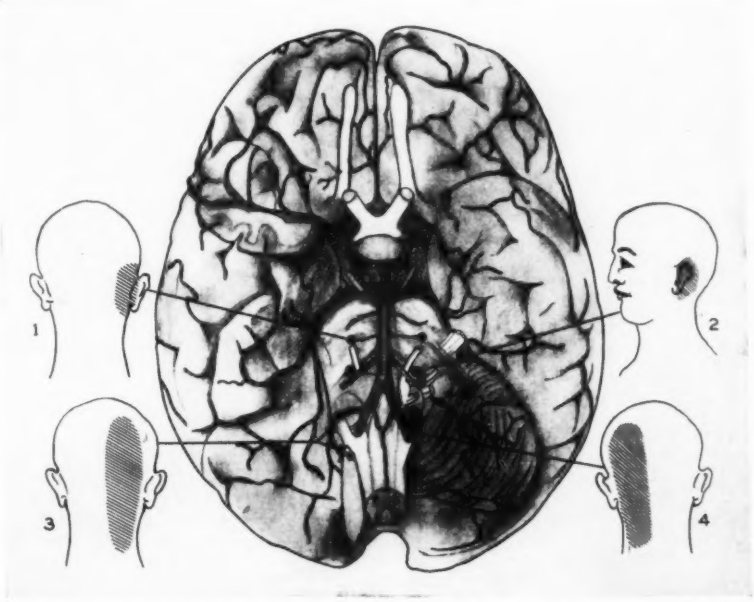

Fig. 6. Arteries at the base of the brain. O indicates the point of stimulation without pain. ● indicates the point of stimulation causing pain.

monly observed bedside fact that a certain number of patients with diseases within the head and headache have that headache augmented by sudden shaking or jolting of the head. This little device, about as big as a match box, is held firmly between the teeth, and the subject is instructed to "jolt" his head, by a sudden turn. There is a smoked glass upon which a stylus writes expressing in G units the force of the displacement of the head.

Fig. 7. The aqueduct of Sylvius, the fourth ventricle, the choroid plexus, and the cisterna magna. O indicates the point of stimulation without pain.

Under these circumstances, it is possible to ascertain how much jolt force is necessary for headache to be induced or augmented. If there is inflammation within the head or traction upon some pain sensitive structure, headache will be induced at much lower amounts of jolt force.

Figure 12 shows what I mean. A youth came to us with severe headache and a story that he had been working with dead mice in his biology laboratory at school. We found that he had an increased number of white cells in the spinal fluid which led us incorrectly to infer that he had a benign lymphocytic meningitis. It took very little jolting of his head to produce a headache, as you can see in Figure 12. Only a small jolt caused enough displacement of pain sensitive structures to cause pain.

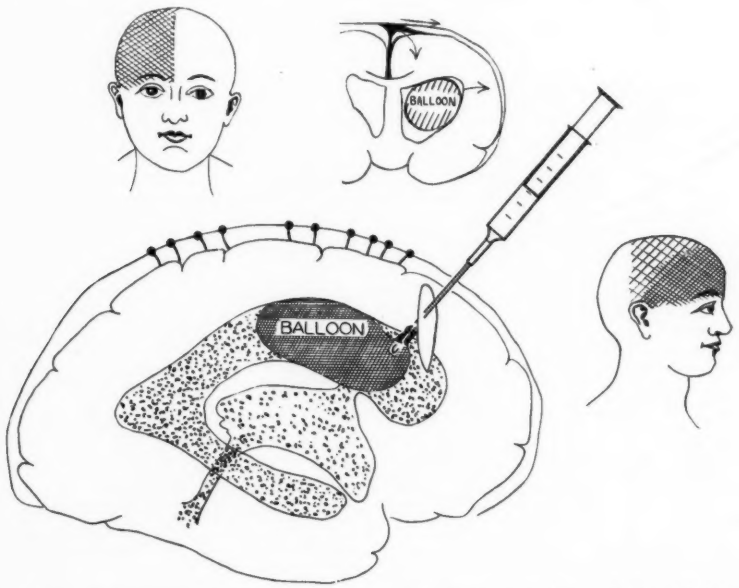

Fig. 8. Schematic representation of a balloon in the lateral ventricle and the tributary veins passing from the cerebral cortex to the superior sagittal sinus. O indicates the point at which the tributary veins were put on traction, causing pain with both inflation and deflation of the balloon.

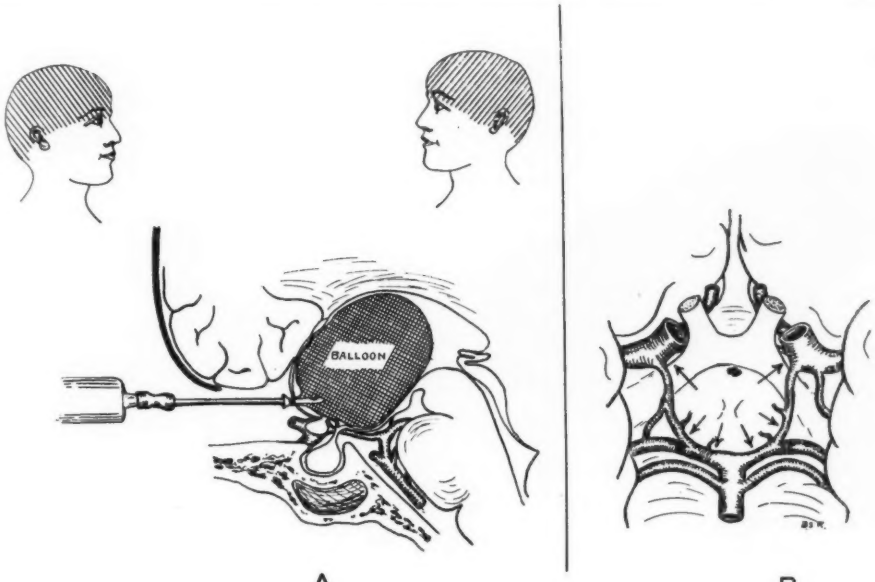

Fig. 9. Schematic representation of (A) an inflated balloon in the third ventricle, and (B) the resulting traction on pain sensitive arteries of the circle of Willis.

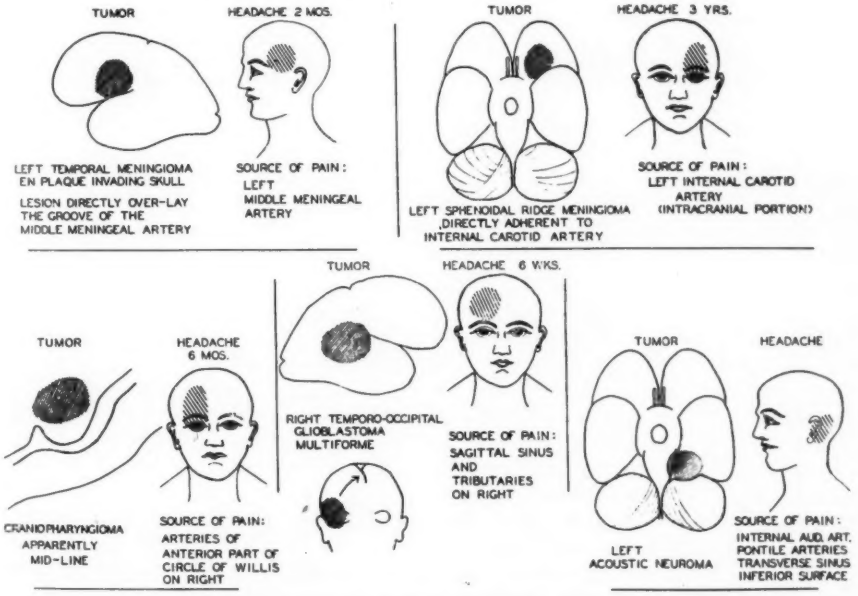
TUMOR
HEADACHE 2 MOS.
LEFT TEMPORAL MENINGIOMA EN PLAQUE INVADING SKULL
LESION DIRECTLY OVER-LAY THE GROOVE OF THE MIDDLE MENINGEAL ARTERY
SOURCE OF PAIN: LEFT MIDDLE MENINGEAL ARTERY
TUMOR
HEADACHE 3 YRS.
LEFT SPHENOIDAL RIDGE MENINGIOMA ,DIRECTLY ADHERENT TO INTERNAL CAROTID ARTERY
SOURCE OF PAIN: LEFT INTERNAL CAROTID ARTERY (INTRACRANIAL PORTION)
TUMOR
HEADACHE 6 WKS.
RIGHT TEMPORO-OCCIPITAL GLIOBLASTOMA MULTIFORME
SOURCE OF PAIN: SAGITTAL SINUS AND TRIBUTARIES ON RIGHT
TUMOR
HEADACHE 6 MOS.
CRANIOPHARYNGIOMA APPARENTLY MID-LINE
SOURCE OF PAIN: ARTERIES OF ANTERIOR PART OF CIRCLE OF WILLIS ON RIGHT
TUMOR
HEADACHE
LEFT ACOUSTIC NEUROMA
SOURCE OF PAIN: INTERNAL AUD. ART. PONTILE ARTERIES TRANSVERSE SINUS INFERIOR SURFACE
LOCAL TRACTION HEADACHE IN PATIENTS WITH NORMAL INTRACRANIAL PRESSURE

Fig. 10. Local Traction Headache in patients with normal intracranial pressure.

Eventually a subdural hematoma was diagnosed and removed, and thereafter the jolt force in G units required to induce a headache was raised to a normal level. The lowering of the jolt threshold of pain for intracranial structures usually means, therefore, that pain sensitive intracranial structures are involved when there is a space occupying lesion in the head, such as a hematoma or a brain tumor.

Fig. 11. Instrument for recording force of head jolt in "G" units.

A patient with increased intracranial pressure and brain tumor further illustrated this mechanism of headache by distant traction and displacement, via an internal hydrocephalus and a cerebellar "pressure cone" on the sagittal sinus and its attachments, the arteries at the base of the brain and the adjacent structures. Lowering the cerebrospinal fluid pressure increased headache, indicating that traction and displacement were the reasons for headache, more than any increased pressure per se.

There is, however, the headache that is asso-

VARIATION IN THRESHOLD TO "JOLT" HEADACHE —

IN A PATIENT WITH LEFT TEMPERO-PARIETAL SUBDURAL HEMATOMA

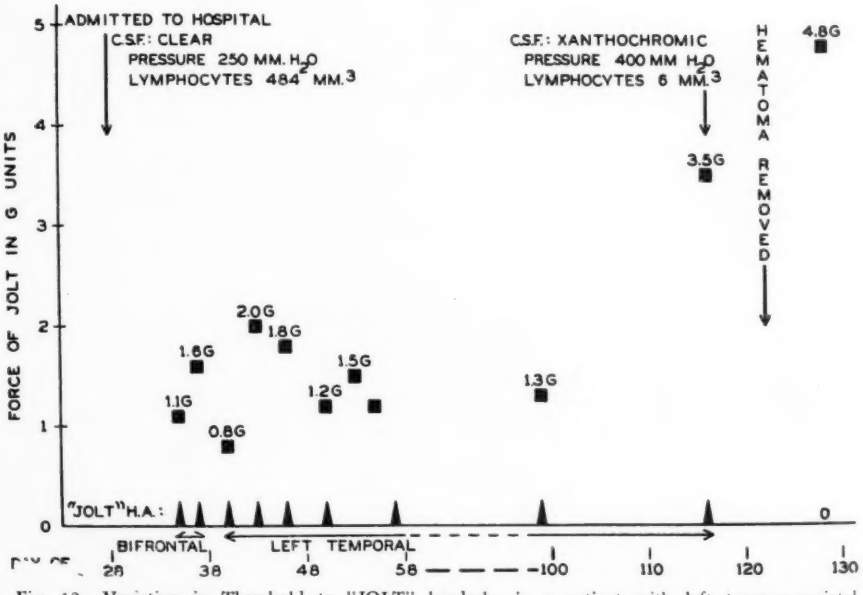

Fig. 12. Variation in Threshold to "JOLT" headache in a patient with left tempero-parietal subdural hematoma.

ciated with lowering of the intracranial pressure, such as the headache that occurs after a spinal puncture or after spinal anesthesia. When an individual was in the upright position, the venous pressure in the longitudinal sinus was somewhere near minus 165 millimeters of water. The pressure in the adjacent subarachnoid space would be approximately the same. When this individual without headache was tilted fifteen degrees toward the horizontal, the pressure in both spaces would become zero. If the individual was placed in the horizontal position, the spinal fluid pressure would become positive or plus 150 to plus 180, the pressure on the right side of the heart. Even with these variations in intracranial venous pressure and spinal fluid pressure, no headache occurred.

When a spinal puncture was done and about 20 cc. of fluid drained off, the pressure in the intracranial venous system remained the same. However, the pressure in the adjacent subarachnoid space became more negative, in the range of minus 215 millimeters of water. In a child with an open fontanelle, the fontanelle is concave after a spinal tap. Now, in tilting a person who had a post-lumbar puncture headache 15° to the horizontal, the headache persisted even though the spinal fluid pressure was only minus 133, because these thin wall veins were distended, and hence gave rise to pain. Therefore it was not the absolute levels of pressure, but again the traction and displacement of the sensitive venous structures in the head that caused headache. One may quickly counter by saying, "I know of patients who have only one or two drops of spinal fluid drawn off in a test tube and who have intense headache afterward." The answer is that much larger amounts of fluid may leak through the hole after the needle is withdrawn, and since the spinal fluid is produced at a rather slow rate, there may be a time lag until the lost fluid is restored.

In summarizing these observations I repeat the five basic mechanisms for headache from structures inside the head that have already been enumerated. First, traction on the veins that pass to the venous sinuses from the surface of the brain and displacement of the great venous sinuses; second, traction on the middle meningeal arteries; third, traction on the large arteries at the base of the brain and their main branches; fourth, direct pressure by tumors upon the fifth, ninth and tenth cranial nerves and the upper three cervical nerves; and fifth, inflammation in or about any of these structures as well as of the pia and brain. It should also be emphasized that the brain tumor headache is the rarest of headaches and in numbers comprise no more than 1 per cent of all kinds of headaches. But when these headaches do occur, although numerically unimportant, they often call for heroic and dramatic measures to obtain relief from pain, and even to save life. I would also call to your attention that infections and structural anomalies of the eyes, the nose, the sinuses and the teeth, dramatic and troubling though they be to the average specialist, nevertheless constitute in numbers a small proportion of the causes of headache, as I am sure most of you recognize.

There remains, then, this major group of ninety plus per cent of headache patients that have pain from noxious stimulation that is in a different category. I turn, then, to the general category of vascular headaches, so far barely touched upon.

Some years ago, it was discovered that if one inadvertently injected a small amount of histamine phosphate into a person he had a headache which lasted five or ten minutes, and which in many ways resembled "bedside" headache yet left him quickly with no untoward effects. We then used this headache for experimental exploration and therefore today when I use the phrase 'histamine headache' I mean a headache which has been induced experimentally for the express purpose of studying some of the facts concerning the natural history of vascular headache.

As of this afternoon, say at 3:25 P.M. no one has proven that there is a clinical histamine headache. By perhaps at 6:00 it may have been done, but right now no one has. Therefore, these experimental procedures have nothing to do with the desensitization against histamine effects which is used in some clinics.

If one injects histamine phosphate one pro-

duces a headache all over the top of the head which is pulsatile and throbbing in quality, and lasts only a few minutes. If one tries to analyze this headache, certain interesting things present themselves. We put a needle in the lower back, amplified the cerebrospinal fluid pulsations through an air system and recorded them on a moving film in a camera. We likewise recorded temporal artery pulsations by means of another air system of amplification.

Immediately following injection of histamine, there is a drop in blood pressure, which quickly returns to normal. It is at this point that headache begins. During this experimentally induced histamine headache, the amplitude of pulsations, both intra and extracranial, was greatly increased, subsiding to control levels with termination of the headache. It was concluded that the increased amplitude of intracranial pulsations after injection of histamine actually represented an increase in the stretch of dilated intracranial vessels with each cardiac systole. This conclusion becomes particularly significant when it is recalled that dural vessels, dural sinuses and the larger pial vessels are important pain sensitive structures.

The question must be in your mind: Perhaps the histamine directly stimulates end organs having to do with pain. If that be so, histamine headaches should be most intense when the amount of circulating histamine is greatest, in other words, during an infusion period. Therefore, we infused histamine for 12 minutes, but there was no headache during that period, although there was a maximum histamine content and a falling blood pressure. During the infusion there was not much change in the amplitude of intra or extracranial pulsations. When the infusion was stopped, however, the blood pressure returned to its normal level and exceeded it for a brief moment. There was a coincident rise in the amplitude of the pulsations and headache began. Thus, amplitude of pulsations increased and pain began with diminishing concentration of circulating histamine, and when the blood pressure had returned to normal. It was the full impact of each cardiac systole upon the relaxed blood vessels which directly stimulated sensory end organs in and about their walls, resulting in a flood of afferent impulses interpreted as pain. It was further observed that raising intracranial pressure by means of saline run into the subarachnoid space greatly reduced, or even eliminated, the experimentally induced histamine headace. It was as if increased intracranial pressure acted as a dampener or support, so that the vessels could not be painfully dilated. Furthermore, it has also been demonstrated by my associate, Dr. E. C. Kunkle, that headache induced by intravenous histamine is promptly eliminated by exposure to positive G of 3.0 to 3.6 units

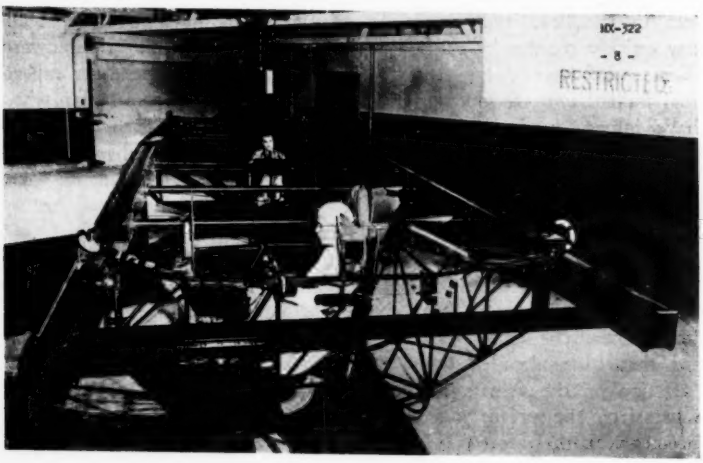

Fig. 13.
Human centrifuge.

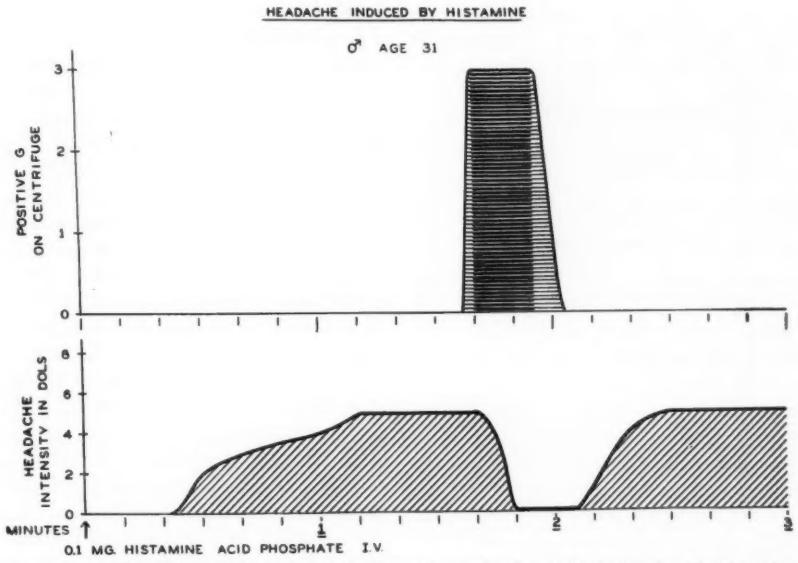

Fig. 14. (Male subject, aged 31.) Elimination of vascular headache induced with histamine phosphate during centrifugation at a positive acceleration of 3.0 g.

(centrifugal force acting in a head-to-foot direction on the human centrifuge (Figures 13 and 14). The decrease in intracranial pressure produced by positive G reduces also the pressure within cranial arteries and thus eliminates pain caused by average blood pressure on relaxed arterial walls.

There are headaches, therefore, of this histamine type which result from intracranial arteries being painfully distended. Such clinically encountered headaches are those that are due to nitrite poisoning, polycythemia, high altitude flying or pressure chamber work, and the fairly common headaches associated with 'hangover,' with hunger and with infections. For example, when we induced a fever in a subject with typhoid vaccine injected intravenously, we demonstrated that intracranial pulsations increased in amplitude, and that headache could be eliminated by raising the intracranial pressure in the manner that I have already mentioned.

When a patient with a fever during a therapeutic attack of tertian malaria shook his head vigorously, and held in his mouth the device described above to measure the force of the jolt in 'G,' it was found that he had a very low 'jolt' threshold. This is another experiment which indicates that pain sensitive structures inside the head are involved in fever headache, a variety of vascular headache. My associate, Dr. Kunkle, having observed the effect of positive G on the vascular headache induced by histamine in subjects in the human centrifuge, made further observations in subjects with a variety of headaches: the tight 'band' headache, the experimentally induced hypertonic saline headache, caffeine withdrawal headache, and hunger headache. He made the following observations: Experimentally induced headaches of non-vascular origin arising from compression with a tight band or irritation of the surface tissues by means of injected hypertonic saline, were only slightly reduced in intensity during exposure to 3.0 units of positive G. On the other hand, the 'caffeine withdrawal' and the 'hunger' headaches arising from distention of intracranial or extracranial arteries, were completely eliminated by exposure of the subjects to 2.0 to 3.0 units positive G in the head-to-seat

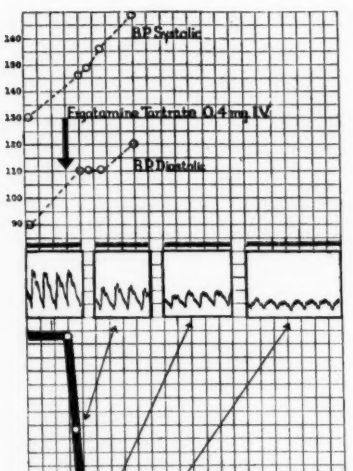

Fig. 15. Relation of the amplitude of pulsations of the temporal artery to the intensity of headache after administration of ergotamine tartrate. The sharp decrease in the amplitude of pulsations following injection of ergotamine tartrate closely paralleled the rapid decrease in intensity of the headache.

direction in the human centrifuge.

It is concluded that headache in these patients just discussed resulted from the sudden increase in vascular pressure within the cranial arteries.

There is another important point I wish to call to your attention, which comes from these last few topics of discussion. When a patient complains of headache associated with constipation he is only telling you that he has another manifestation of a tension state in which both headache and constipation occur. There is no "constipation headache" per se, and there are no headaches resulting from noxious stimulation or disease of structures below the head. In other words headaches do not occur as referred pain from other parts of the body. Such concepts are now quite obsolete.

Sir Henry Head believed if certain parts of the chest and abdomen were stimulated, as in disease of certain organs, that referred pain in localized areas of the head would result. However, the patients he described with such so-called referred headache associated with endocarditis, tuberculosis and pneumonia also had fever, sepsis and bacteremia. As already indicated to you, fever headache is a vascular headache resulting from distention of pain sensitive arterial walls. Pain from the heart can be referred as high as the lower jaw, but otherwise disease from the chest does not give rise to pain in the head.

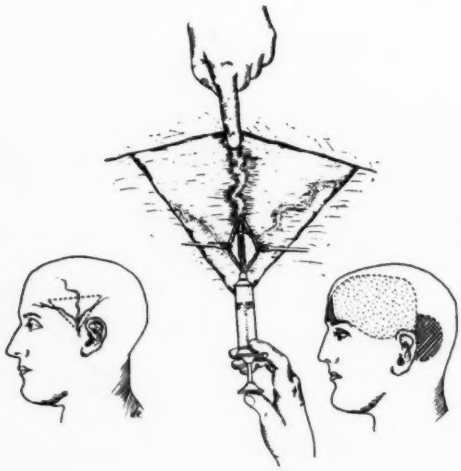

Fig. 16. Head pain caused by distention of the temporal artery. (Operation was performed by Dr. Bronson Ray and the drawing is included through his courtesy.)

Another instance of headache occurring with increased pressure within the cranial arteries is that following the administration of adrenalin. Such headache occurs in the rare instances of adrenal tumor when by inadvertently massaging the tumor, too much of the individual's own adrenalin is squirted into his blood. But again, this is not referred pain, and the resultant headache is due to sudden dilatation of pain sensitive structures within the head. Nor should such headaches be confused with the headache associated with hypertension. People with high blood pressure often have headaches. However, only half of them do, and they do not have higher levels of blood pressure than the half who have no headaches. Often enough, the blood pressure gets higher as the headache disappears, as illustrated in Figure 15 where you see elimination of headache in a subject following administration of the vasoconstrictor agent ergotamine tartrate. This man's headaches frequently occurred when his blood pressure was lowest. The headache of essential hypertension is very much like migraine headache and has a great deal to do with the state of the cranial vessels, and the life situation and feeling state of the patient.

Let us move on to consideration of the headache that arises mainly from the blood vessels on the outside of the head. If we explore these vessels with electrodes in the same way as we have the vessels on the inside of the head we find they are all exquisitely sensitive wherever they are stimulated. There are other pain sensitive structures on the outside of the head but the arteries are especially sensitive.

Figure 16 shows that these vessels not only are sensitive when stimulated with electrodes but that they hurt very much when they are distended. For example, if you put a needle attached to a syringe into one of the temporal arteries, compress it a short distance away with your finger and then push the plunger of the syringe to dilate that portion of the artery, the subject immediately experiences pain.

If you rhythmically dilate the vessel in this way the patient has throbbing pain. Now, there are a certain number of people who have inflammation of the arteries on the outside of the head, i.e. cranial arteritis. These are exquisitely sensitive arteries, and headache is, indeed, terrific. Re-section of the involved artery will immediately eliminate the headache. But, there are many, many headaches involving these vessels that are *not* modified for any length of time by any such radical surgical procedure. The individual soon finds that there are many other blood vessels in the head that can dilate painfully and that surgery, at best, can give only temporary and short relief. Therefore, let us waive, at once, all consideration of the surgical approach to vascular headache.

Figure 17 shows what a man looks like who is having a migraine headache. This is a common type of headache, perhaps numerically the commonest, and comprises between sixty to

Fig. 17. Appearance of the temporal artery before and after termination of migraine headache by ergotamine tartrate.

Figure 18

PEDIGREES ARE FROM 119 MIGRAINE PATIENTS
48 MALES AND 71 FEMALES WHO HAD
343 RELATIVES WITH MIGRAINE

20 Patients Had No Migraine Relatives
66 Patients Had 1-3 Migraine Relatives
22 Patients Had 4-7 Migraine Relatives
11 Patients Had 8-19 Migraine Relatives

seventy, or even more, per cent of all headaches. Now, how is one to define a migraine type of vascular headache? The word "migraine" comes from the Greek "hemikrania" and through much mouthing and common use the front and back end of the word have been dropped. So that if we follow it historically, we find that the word has been at times "hemigranea," "emigrania," "megranea," "megrim" and now finally, "migraine," meaning "one-sided headache."

The migraine headache can be defined as a periodic and recurrent headache, usually unilateral in onset, but commonly becoming generalized before the episode is ended, often associated with nausea and vomiting, and preceded in about five to ten per cent of instances by visual disturbances. It commonly runs in families, Figure 18, and the headache is dramatically modified by vasoconstrictor agents.

Now, to repeat, Figure 17 shows a man having a migraine headache. I think you can see in the picture on the left that the individual's temporal arteries stand out clearly. If he were here in person you would also see that his skin readily pits, that it is slightly edematous. You would also be able to see that his eyes are effused and he tears readily, and that his nose on the left side is occluded, and yet with profuse watery secretions running out of it.

The patient complains bitterly of the throbbing pain on the left and he feels as if he were sick to his stomach. The light 'hurts' his eyes.

Now, the picture on the right shows the same individual in approximately the same light and circumstances; his temporal arteries no longer stand out, his tissues are less edematous and he is free of headache and only twelve and a half to fifteen minutes later.

Figure 15 shows what happened during this time. The black at the bottom indicates the intensity of the headaches. The next line represents schematically what happened to the amplitude of pulsations of the temporal artery when

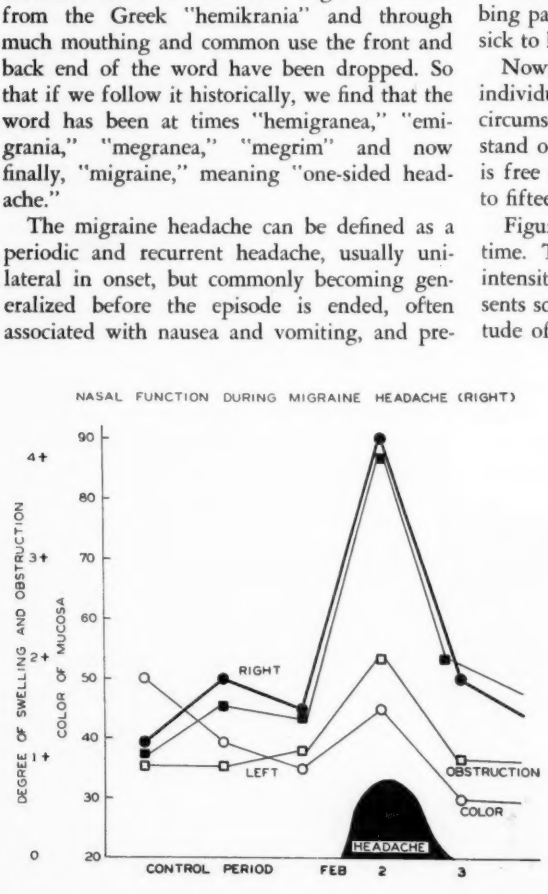

Fig. 19. Hyperemia and obstruction in the nose during migraine headache attack; vasodilatation in the nose accompanying distention of cranial arteries.

a vasoconstrictor agent such as ergotamine tartrate was given intravenously. There was then a sharp fall-off in the intensity of the headache. Were this agent to be given intramuscularly, it would take forty-five to sixty minutes to operate, but essentially the same relationship could be demonstrated between decrease in the amplitude and fall-off in the intensity of headache.

Let me emphasize that other vasoconstrictor agents will do essentially the same thing, but therapeutically they are far less practical. Consider levophed, (which is nor-adrenaline) for instance. As the blood pressure is raised by its vasoconstrictor action, the subject's vascular headache of the migraine type diminishes, as does the amplitude of pulsations of the temporal artery.

Now, it happens that the temporal artery is the one most commonly involved in this kind of headache but any artery on the outside of the head may be the main source of pain. Indeed, I would be extremely bookish, were I to indicate that only the vessels on the outside of the head are involved. There is also evidence that vessels inside dilate, even though they may not be painful during such an attack; suffice it to say that manipulation of the outside vessels markedly reduces the intensity of the pain and terminates the headache attack.

Any agent that will constrict these dilated arteries will modify the intensity of the pain, thus relieving the subject.

Well that is not the whole story, unfortunately, because I think you have probably seen many people after exercise on a warm day, or merely sitting in the sun, with dilated cranial arteries, without a headache. After you yourself have been immersed in a hot tub of water, your cranial arteries would be outstanding like a snake and yet you would have no headache. So there must be another factor in headache in addition to dilated cranial vessels and I will tell you about that later in this talk.

Intensity of a vascular headache of this sort can be lessened by direct mechanical pressure. Thus, you often see people with headaches sitting with their hand pressed against the side of their aching head. Unfortunately the vessels are not completely occluded and soon they are thumping away painfully again. Besides the tissue pressed upon is very tender, and the pressure itself may hurt.

Now, that tenderness is the clue which I hope to explore with you a little later.

If you take a person having a migraine headache, assuming you can persuade him to do this disagreeable thing, and spin him on a human centrifuge, you can spin out his headache for a short time and sometimes it doesn't come back, and sometimes it does, which indicates that again, the vascular structure is involved and that the intra-vascular pressure is pertinent to the occurrence of the headache.

Now, there are a few "Don'ts" or errors of judgment about headaches that should be mentioned. There are so many people who have occlusion of one or both nostrils during their vascular headache attacks that they are apt to infer that the nose is the prime cause of the trouble, and that if they only had their noses treated, the headache would not occur.

All I can say is that the mucous membranes of the turbinates, the linings of the nose and the para-nasal spaces innocently become involved in this cranial vasodilation with swelling, redness, occlusion and increased secretion without being causative factors in the headache. (Figure 19).

Not only the big vessels, but the little ones participate in the attack. Dilatation of small vessels is not painful in most instances, but to some degree it is. In the case of the eye, the pain and burning in the eye that one experiences during vascular headache is related to the vasodilatation. It is not the primary cause of headache however, but it is merely a concurrent manifestation.

If you examine the two eyes during a unilateral migraine headache the conjunctival vessels will be seen to be dilated on the side of the headache. (Figure 20.) In addition there is edema, and the light hurts and the individual rubs his eyes, saying that it burns. Again, this is not a primary cause of headache.

Figure 20 brings us to that phenomenon of the migraine attack which is experienced by only a few. I refer to that part of the attack

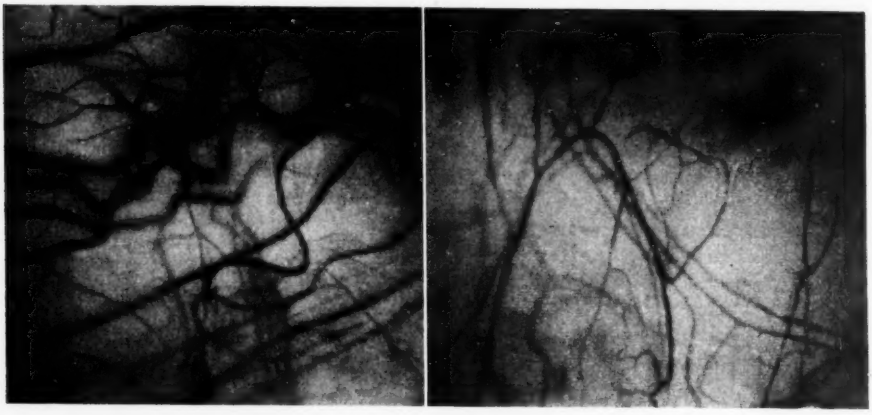

Fig. 20. Contrast in appearance of vascular structures in right and left bulbar conjunctivae during right-side headache.

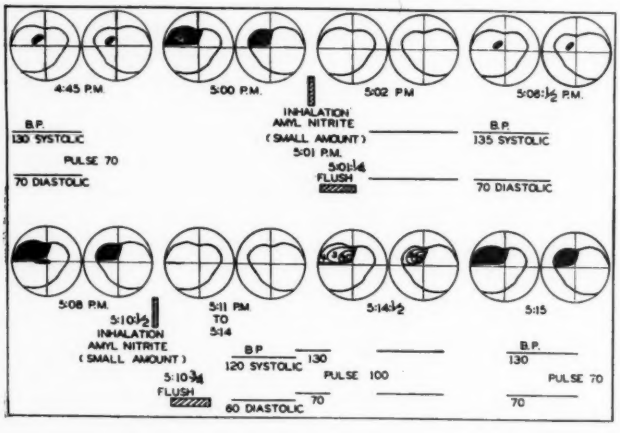

Fig. 21. Effect of inhaling a small amount of amyl nitrite on pre-headache scotomas in a subject with migraine.

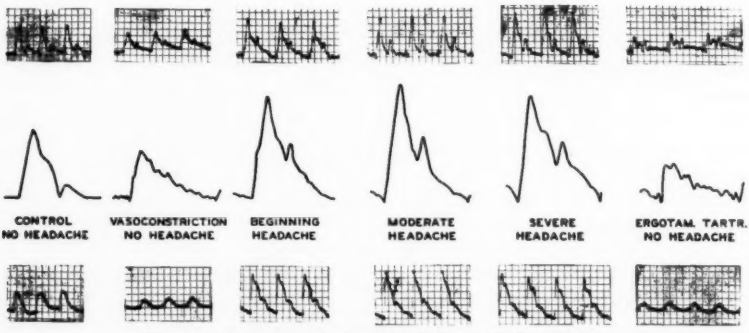

Fig. 22. Representative Temporal Artery Pulse Wave Contours in Two patients during vascular headache.

which actually precedes the painful part of the attack by ten or fifteen minutes, usually. This is described as a visual scotoma, and this particular person, a physician, was able to plot his visual field effects, as they developed. He came to the laboratory at 4:45 P.M. when he could see the beginning of a blind spot or scotoma which had increased in size by 5 o'clock. (Figure 21).

Now, the inhalation of a small amount of vasodilator such as amylnitrate immediately cleared up the visual defect, only to have it recur in four minutes and be re-established in six minutes.

The inhalation of this minute amount was repeated and again the visual fields cleared up, only to be impaired in the original way in another six or eight minutes.

What this means to me is that there are vasomotor disturbances of both kinds in the head, as a part of the migraine attack. The non-painful part is a preliminary or concurrent vaso-constriction, eliminated by vasodilators.

The painful part is the vasodilator phase, which comes after the vasoconstrictor phase, and which in turn is modified or eliminated by vasoconstrictor agents. (Figure 23).

Now, some persons by breathing a carbon dioxide and oxygen mixture in this preliminary phase, can abort the subsequent, painful portion. Some persons never go into the painful portion, but merely have the vasoconstrictor component.

Mind you, less than 10 per cent of those who have migraine attacks have this vasoconstrictor phase so conspicuously as to give rise to scotomata but most all people look very pale before an attack, and when the headache starts, may appear markedly flushed.

Figure 22 illustrates the various kinds of pulse waves to be recorded from a cranial vessel involved in a headache. There are pulse waves during the pre-headache or control period, the vaso-constrictor phase without headache, the beginning headache and the subsequent full development of headache and finally, pulse waves after the headache has been terminated by the vasoconstrictor effects or ergotamine tartrate. A great many variations in the amplitude and in the shape of the pulse waves seem

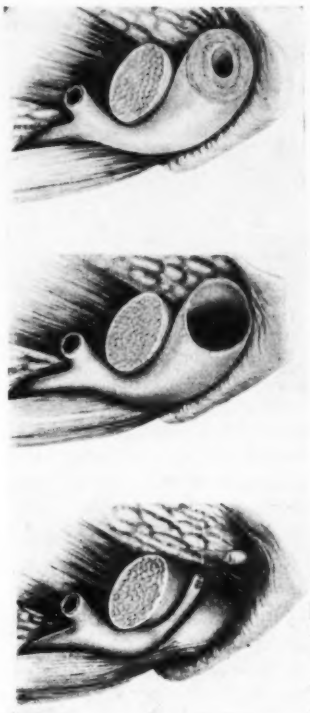

Fig. 23. Schematization of phases of constriction, dilatation and edema of a cranial artery during a migraine headache attack.

to be a feature of such subjects, as compared to non-headache subjects. It is as though they used the cranial vaso-motor apparatus in an inappropriate way in response to a variety of situations.

I would like to call your attention to a very important fact, namely, that every one who has to do with the therapy of human beings becomes involved in the headache problem.

If you are a dentist, you are taken up with many headaches that have to do with blood supply of the upper and lower jaw, and you see how often the temporomandibular region becomes involved in vascular headaches.

If you are a rhinologist, you are apt to blame a great many headaches upon the nose or paranasal spaces. If you are a neurologist, you have to think of the possibility of expanding intra-

Fig. 24. Four views of a hematoma about the left temporal artery that developed 48 hours after onset of headache in subject M.G.

cranial lesions. If you are an otologist, certain headaches may seem to involve the ear. And, an orthopedist looks at headaches arising from painful dilatation of the occipital artery at the back of the head.

Now, these headaches can be of any intensity, any duration, for they can last a few minutes to twenty-four hours, or a few days, and with any kind of periodicity, they can occur regularly or only in certain seasons or they can occur in clusters, with or without a predictable timing.

The one thing they all have in common is aching pain, with more or less throbbing. Depending upon what kind of specialist you are, you are apt to use your favorite device in trying to help these patients.

But please allow me to proceed with the argument.

Figure 24 shows a woman who has had a headache for several hours. The unique aspect of her headache is that she has developed a hematoma on the side of her head. It was an egg-shaped lump that lasted for several days. The black and blue spot over the lump can be seen in the picture. It was extremely tender—so much so that the slightest pressure for some distance around the lump elicited pain. That gave us a good opportunity to explore the mechanism of tenderness and its relevance to the headaches of the vascular type of headache.

Surgical re-section of this lump showed that the temporal artery, the one most implicated, didn't actually rupture but there was a collection of bloody fluid as though myraid small vessels might have allowed fluids and many red cells to flow into the tissue.

It looked as though sustained dilatation had been accompanied by a lowered permeability of these small blood vessels.

Dr. Hardy contrived a device to measure the amount of pressure you have to apply to the tissue of the scalp in order to have the individual say that it just hurts. This is a fairly good measurement of pain threshold. (Figure 25).

Figure 26 shows that if you measure the sensitivity of the tissue about the head, that is to say, the deep pain thresholds at the site of the headache both during headache and between headache attacks, you get different pictures. The right side of this chart shows the pain thresholds are rather high when a subject is free of headache. The left side shows the pain threshold is low at the time of the headache. In fact you can barely touch the part without having the patient complain of pain.

Patients with such headaches don't like to have glasses over their ears or a hat on their head, and certainly don't like to have anybody touch their head for any reason.

What is the meaning of this tenderness, and what is its bearing on headache?

The nearer you approach the artery involved in throbbing pain the more sensitive the tissues are and the further away you go the less sensitive they are. Ultimately in some headache attacks the whole head can become pretty sore.

Now look at Figure 27. When you introduce the powerful vasoconstrictor agent norepinephrine, it quickly abolishes the headache. As the headache diminishes, so also, does the sensitivity

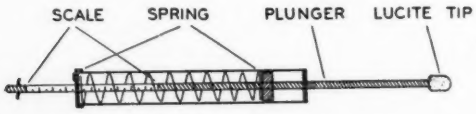

Fig. 25. Diagram showing the princple of the Hardy deep pain dolorimeter.

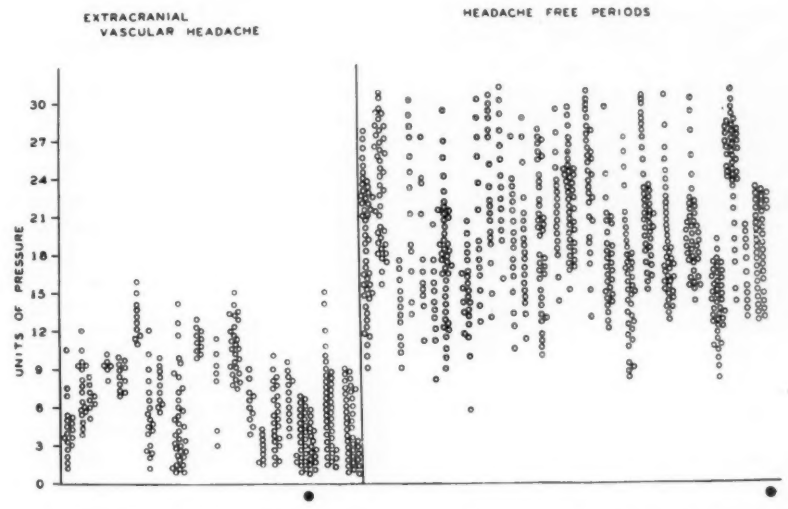

Fig. 26. Deep pain threshold measurements made in the zone of headache during migraine attacks in 10 subjects compared with deep pain threshold during headache-free periods.

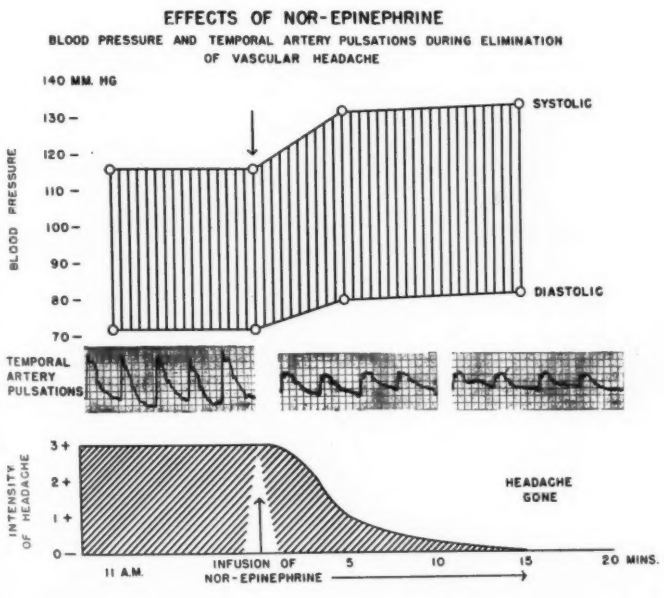

Fig. 27. Effects of Arterenol (Nor-epinephrine) on the blood pressure and temporal artery pulsations during elimination of vascular headache.

of the tissue diminish. That isn't true of the headache which spontaneously diminished. Many people will tell you when their headaches end spontaneously, the scalp is sore for hours and sometimes for days afterward.

This looks as though the norepinephrine did something more than constrict blood vessels. It looks as though it pulled out of the tissue something which was causing it to be tender and to hurt with every cardiac systole. We began to explore the nature of this "tissue juice," and after many observations we have evidence that there accumulates in the tissues during the headache a substance or substances having the capacity to lower the pain threshold.

Chemically, this substance has the properties of a polypeptide, and it may be bradykinine. My view at the moment is that with the dilation of large and small vessels, this threshold lowering substance accumulates in the adjacent tissues and makes them very tender. Now the ordinarily non-painful dilatation of an artery may become exceedingly painful because of the presence of this threshold lowering substance.

Figure 28 carries us into a very striking aspect of headache phenomena. Most people who have vascular headaches of the migraine type will tell you that they pass large quantities of pale urine either at the peak of or shortly after the peak of the headache. Indeed, if you weigh these individuals every day, you will find that they lose two to twenty pounds during an attack, only to gain it slowly back until another attack occurs. And yet, this has nothing to do with the pain mechanism. And modifying the accumulation of body fluid does not alter the likelihood of getting another headache, as is shown in Figure 29.

And now, about migraine headache and how can it be modified? In Figure 30 you will see indicated the intensity and frequency of the headache of a 44 year old woman, with migraine headaches during the previous 7 years.

She was reassured that she did not have a brain tumor or other serious brain disease. She was given placebo pills to take at regular intervals, and she was also strongly reassured that they would "help her." As a result, she was headache-free for about six weeks. So do many patients, who getting reassurance from their physician or their dentist have the frequency and intensity of their headaches dramatically modified for shorter or longer periods. This fact that their state will improve and headache be lessened by means of manipulations of the gall bladder or the teeth, and as well by sugar pills or new glasses points up what I have just been saying. Again, the black lines indicate the almost daily frequency and the intensity of the attack.

On September 1st, a sympathetic doctor listened to this woman's story of her relations with her child. In short, the child hated her and she hated the child. She being the bigger of the two, was in a position to do more harm.

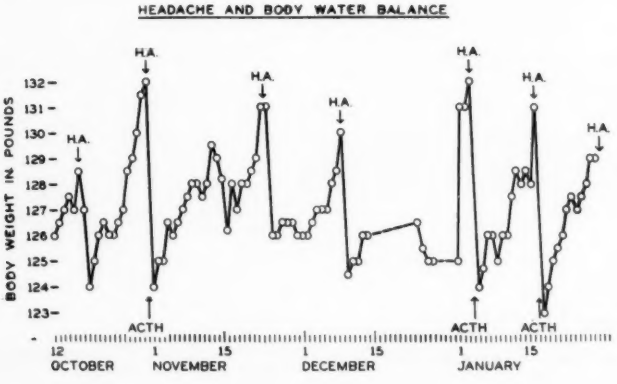

Fig. 28. Observations on the occurrence of headache and the daily fluctuations in body water balance in one patient during a period of 16 weeks.

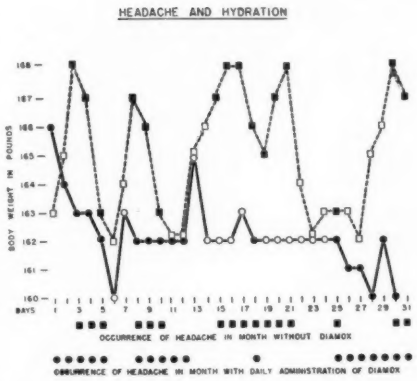
HEADACHE AND HYDRATION
BODY WEIGHT IN POUNDS
DAYS
OCCURRENCE OF HEADACHE IN MONTH WITHOUT DIAMOX
OCCURRENCE OF HEADACHE IN MONTH WITH DAILY ADMINISTRATION OF DIAMOX

Fig. 29. A comparison of the occurrence of attacks of vascular headache in a patient during one month with and one month without the daily administration of the diuretic, "Diamox".

She had actually struck the child, and had frightened herself by the fury of her actions and its implications. A mixture of guilt, resentment, rage and shame was poured out upon a sympathetic ear, and the feeling of relief from this confession and the sympathetic response of her doctor stopped the daily occurrence of the woman's headaches for 10 days. (Figure 31).

On this tenth day when she came back to her physician he was, in her eyes, less sympathetic, and aroused her guilt and her resentment by his utterances. She felt betrayed.

Under these circumstances and as her feelings of anger at being "sold down the river" mounted this woman developed one of her characteristic headache attacks, which was, as you may see in Figure 31, promplty eliminated by the use of a vaso-constrictor agent, ergotamine tartrate. In other words, the facts of the noxious life situation of this woman, reintroduced in an experimental setting, could precipitate the symptom, which symptom could be eliminated by a vaso-constrictor agent that reversed the vascular reaction giving rise to pain. A series of similar observations has contributed much insight and understanding of the complicated nature of these vascular headache attacks.

Figure 32 has to do with the headache associated with the contraction of the muscles of the scalp and neck. Now I call your attention to this extremely common type of headache, which is non-pulsatile, which may go on for days, weeks, or years, and usually is not of great intensity. It ensues from inappropriately using the muscles of the neck and head in meeting day-to-day challenges, frustrations and disappointments. It is the headache you get from driving long hours on a hot summer day in traffic that "gnashing your teeth about" won't overcome. Again the individual feels as if he had a hat on when he has none, that his neck is in a cast, that his jaws ache, that his shoulders are sore, that if he could be rubbed he would feel better; that if he could but stretch his neck he would feel more comfortable. Action potentials recorded from head and neck muscles at such a time indicate vigorous contractions. Pres-

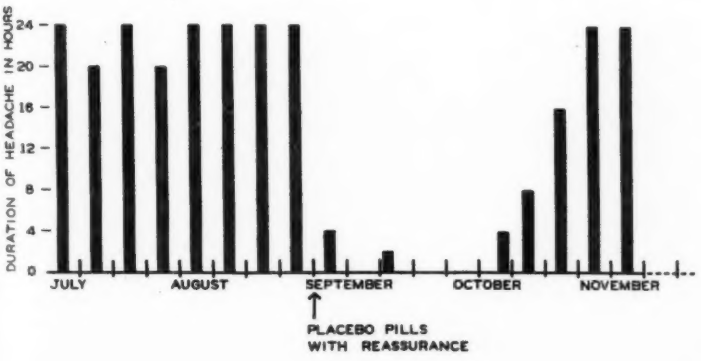

Fig. 30. Record of the headache attacks of a 44 year old woman with migraine during the previous seven years.

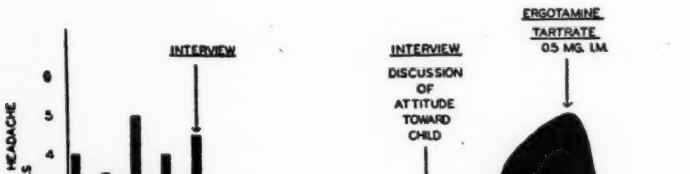

Fig. 31. Experimental precipitation of a migraine headache attack in a setting of mounting tension by the introduction of a topic arousing conflict, anger, and guilt.

sure pulse waves of the relevant cranial arteries measured at the same time show a marked diminution in amplitude, indicating local vasoconstriction with reduced blood flow and muscle ischemia as a consequence. Pain thus ensues partly as a result of sustained muscle contraction and partly as a result of local tissue ischemia. This is the kind of reaction that occurs in an individual who is "cramming" for examinations, looking into microscopes for long periods, or reading books for hours on end. Such tension may be more responsible for pain than excessive use of structures in the head. For example, one man had headache of low intensity for a long period. He worked at a job he did not like, surrounded by companions who gave him no friendship, he was married to a woman who was outspokenly disappointed in his performance. Without friends or companions or hobbies, without affection or approval, he had come to be a listless, disappointed, resentful, frustrated man, "getting nowhere," "fed up,"

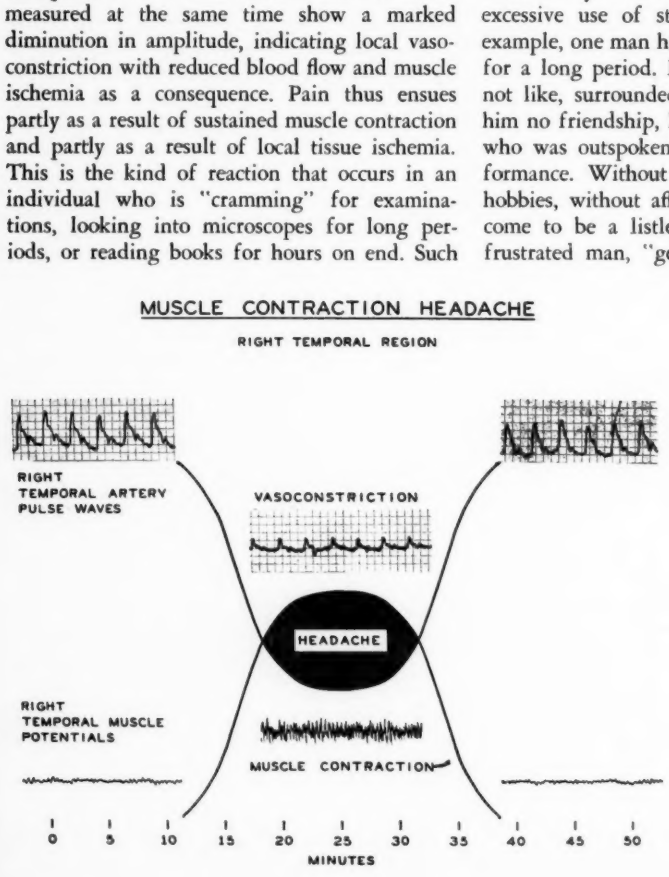

Fig. 32. Muscle contraction headache, right temporal region.

and angry. He had a headache day in and day out. It was possible in his case to affect his state considerably. He had an opportunity to obtain other work; his wife could be induced to take a more sympathetic attitude; he joined a club of people he liked and who afforded him friendship, and with the use of a small amount of sedative, three weeks later he was more comfortable, the action potentials recorded from his muscles had diminished, and he was headache-free (Figure 33).

Sustained contraction of skeletal muscles about the head may be compared, in other patients, to the contraction of the abdominal wall over an inflamed peritoneum. About thirty percent of headaches fall into this general category, with or without concurrent vascular components. It is also a secondary effect of inflammation in the head, or may occur independently of any inflammation during sustained alertness and tension in an individual who is prepared biologically to spring into action, but which action never comes off.

There is a numerous and very troublesome group of patients who have headache following head injury. Such an individual was studied at New York Hospital, a typical "G.I." who was injured in Guadalcanal during a bomb burst. He had been "pushed around from pillar to post"; he was disturbed, angry, feeling abused. Because of his sustained headache he was sent from veterans' hospital to veterans' hospital, from clinic to clinic. Action potentials recorded from his cranial musclature indicated excessive muscle contraction. He had a tender point in his scalp which when pressed upon, increased the intensity of his headaches. We injected a small amount of procaine around this tender area, the action potentials were reduced, and, for a while, the patient was more comfortable. We observed the same fellow on days when he was relatively comfortable and without painful sensations in his head. Then action potentials from his cranial muscles were minimal. But on days when he "nursed his grudge" his head not only felt "tight and sore" but action potentials were high.

Occasionally in an individual following head trauma one can find a painful scar or neuroma which may cause head sensations for weeks or years. These constitute a minor group of "post-traumatic headache". More commonly, individuals with "post-traumatic headaches" have ordinary vascular headaches or muscle tension headaches.

May I call your attention to observations in regard to the nose and the upper airways: it is impossible to ascertain from the localization of pain what specific part of these airways or their coverings might be involved. For example, putting a pack under the inferior turbinate causes a burning pain locally, followed by an

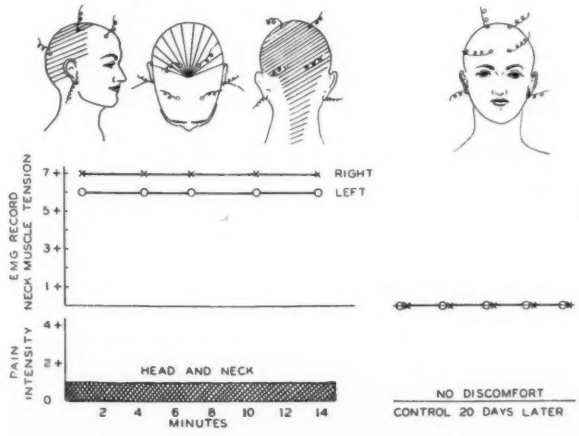

Fig. 33. The effect of massage upon muscle contraction and pain in the neck.

Fig. 34. Lines lead from the points stimulated in the nasofrontal duct to the areas in which pain of 5 dols intensity was felt.

aching pain accompanied by a reddening of the cheek and inability on the part of the subject to tell whether pain is coming from his teeth, his eye, his frontal sinus, his antrum, or perhaps his nose. Localization of pain is poor in the general area of distribution of the first, second and third divisions of the fifth cranial nerve. If we now stimulate various structures in the airways with a faradic current of intensity sufficient to cause a one plus* pain on the tongue, we find that the nasal and paranasal structures have a varying degree of pain sensitivity. Thus, faradic stimulation of the turbinates induced a 4 to 6 plus pain experienced in the upper teeth, the frontal region, along the supraorbital ridge, over the zygoma and in the temple. A pain intensity of 5 to 8 plus was reached when this same faradic stimulus was applied to the nasofrontal duct (Figure 34) but once inside the frontal sinus the unchanged intensity of stimulus induced only a 1 plus pain, similar to that induced on the top of the tongue. The ceiling of pain intensity of 9 to 10 plus is experienced on stimulation of the ostium. However, the walls of the maxillary sinus are not much more sensitive than the septum or the tip of the tongue (Figure 35). In one patient it was possible to put a catheter into an antrum, through an opening left after a faulty tooth extraction. A balloon was attached to the end of the catheter entering the antrum. When this balloon was inflated to 50, 80 or 100 mm. of mercury pressure, no pain ensued. However, when a pressure of 80 mm. mercury was maintained for 2 to 3 hours, a 1 plus pain developed which was experienced in the upper teeth, and along the area of the zygoma, radiating back toward the ear. Forced inhalation of cold air increased the intensity of pain to 4 plus. Procainization of the swollen, engorged turbinate and the nasal entrance of the ostium eliminated the pain. Similarly, after plugging the ostium on the nasal side and then inducing a negative pressure of 100 mm. of mercury, the cotton plug was pulled into the ostium, the ostium walls were irritated, and immediately a 4 plus pain was experienced in the side of the nose and in the teeth.

* The subject's estimated "plusses" on the basis of 10 plus being the worst pain they could imagine from past experience.

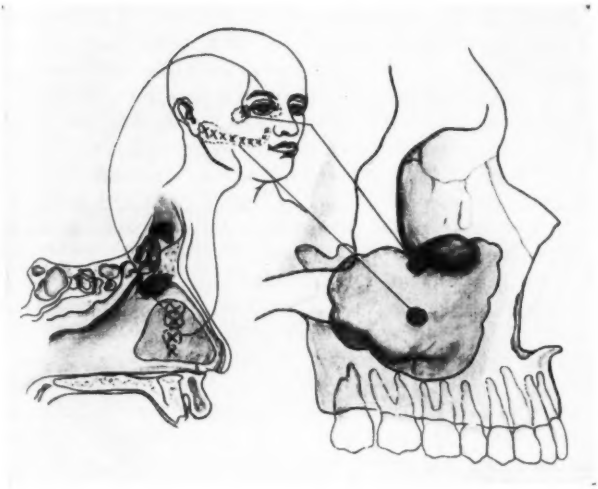

Fig. 35. The points stimulated on the septum are shown by crosses and on the lateral wall of the maxillary sinus by cross hatched circles.

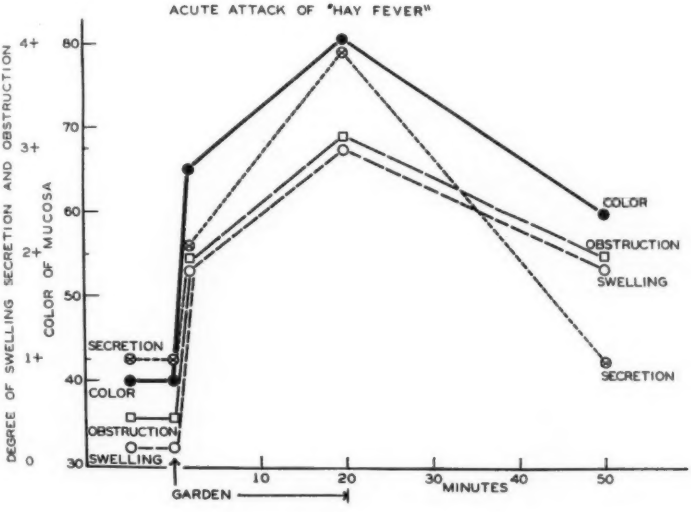

Fig. 36. Hyperemia, Hypersecretion and obstruction in the nose during an abortive attack of "hay fever".

The lesson from these observations is well known to all of you; that much of the pain from the nose comes from the *approaches* to the nasal and paranasal spaces and rather less from the paranasal spaces themselves. If these spaces are diseased, obviously they must be treated, but as sources of headache they are indeed much over-rated. In our time the word "sinus" has become the equivalent of "headache" in common language. But indeed we have found inflammation in the sinuses themselves to be a rare cause of headache.

There are important facts concerning the nose, namely, the capacity of the nose to react to a number of environmental stimuli, chiefly in the nasal rather than in the paranasal spaces. A simple experiment was performed in which the subject held before his nostrils some smell-

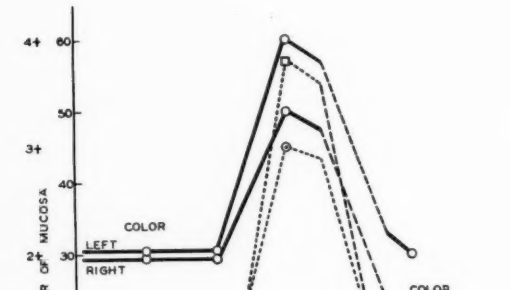

Fig. 37. Hyperemia, hypersecretion and swelling of the nasal structures during weeping; the reaction of shutting out and washing away.

ing salts and inhaled the irritating amonia fumes. We recorded the color, swelling, and amount of secretion and obstruction before and after the inhalation. Not only did the nasal mucous membranes become reddened and swollen, but there was profuse secretion and marked obstruction to breathing. The eyes were red, swollen and wet. The diaphragm stopped moving and the individual could not breathe and looked and acted as though he were provisionally shutting out a noxious substance. The nose was promptly restored to normal appearance and function on removal of the ammonia fumes. Another sensitive subject went into a rose garden where the rose pollen induced a similar shutting out and washing-away reaction. (Figure 36).

Suppose now you have before you a woman who has had a lifelong trouble of nasal difficulties of pain and obstructions and who has had numerous operations on the nose. In this case, instead of presenting a noxious stimulus in the form of a chemical agent or a gas, we introduce a *topic* having to do with her relations toward her husband and her husband's unsympathetic attitude toward her nasal troubles. Under these circumstances there is a similar reaction of reddening, swelling, hypersecretion and obstruction as she burst into tears.

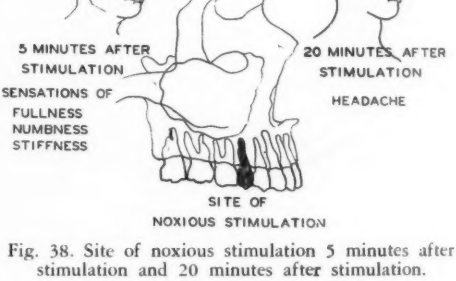

Fig. 38. Site of noxious stimulation 5 minutes after stimulation and 20 minutes after stimulation.

(Figure 37). Such a reaction may be of short duration or long-lasting.

For a man, the shedding of tears is less common. Under the circumstances of partially expressed rage and desperation in an interview in which topics having to do with a male subject's personal life were brought into focus, his nasal mucous membranes reacted as if he were shutting out and washing away a noxious gas, dust, or chemical agent. He declared himself to be on the verge of tears as he discussed his unattractive marital partner and his unpleasant life situation. Such a reaction in an unhappy life situation however, may be long-

lasting and become the soil for secondary infections resulting in pain and chronic inflammation in the nasal and paranasal spaces.

Consider, for example, one of our young house officers, long a reactor with his nose under circumstances of trouble, who found himself associated on his ward with another physician exceeding himself in age, but junior in other ways, including his sense of responsibility, his medical knowledge and judgment. Our subject was responsible for the conduct of his ward and the welfare of his patients. His older assistant performed his duties carelessly and took suggestions with resentment and contempt. Relations between these two were strained, yet our young house officer could not express himself nor complain to his seniors. Before this man had come in contact with this irritating fellow, this "noxious agent" in his environment, daily observation of his nose had revealed a quite normal appearance and function. During the two weeks when he wished he could "shut out and wash away" the poor performer whom he considered to be a threat to his own success, his nasal mucosa were red and swollen, secretion was profuse, obstruction to breathing bothersome, and headache frequent. As soon as this "noxious agent" was removed from our young man's environment, his nasal difficulties and his headaches were eliminated. This man's symptoms had closely resembled those associated with infection. Similar complaints in patients are numerous. Their condition constitutes a physiological reaction, but successful treatment will rest largely upon their conviction of their doctor's interest in them and concern about the life circumstances which engendered their adverse nasal reaction.

May I call your attention again to the reddening of the cheek which accompanied the irritating effects of noxious stimulation of the turbinates. This was demonstrated to be an area of secondary hyperalgesia. That is to say, a pin prick is felt sharper in the area, although we have established that there was no change in the pain threshold. In several such observations, the cheek got redder, the eye got red and watered. This was due to a heightened central excitatory effect of the noxious stimulation, and not to a local liberation of an agent in the skin. When procaine was applied to the irritated turbinate, all of the effects, including the hyperalgesia disappeared.

The same phenomenon has been demonstrated in a person who had a headache associated with a diseased tooth. When procaine was injected in the area of the headache and hyperalgesia, there was some reduction of pain, but it was completely eliminated only when the procaine was infiltrated about the diseased

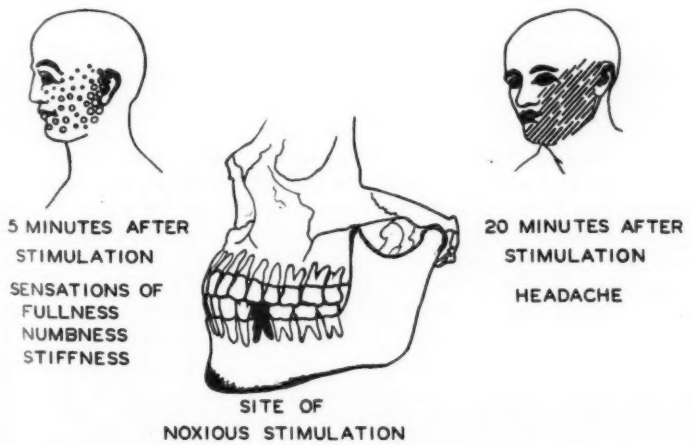

Fig. 39. Site of noxious stimulation 5 minutes after stimulation and 20 minutes after stimulation.

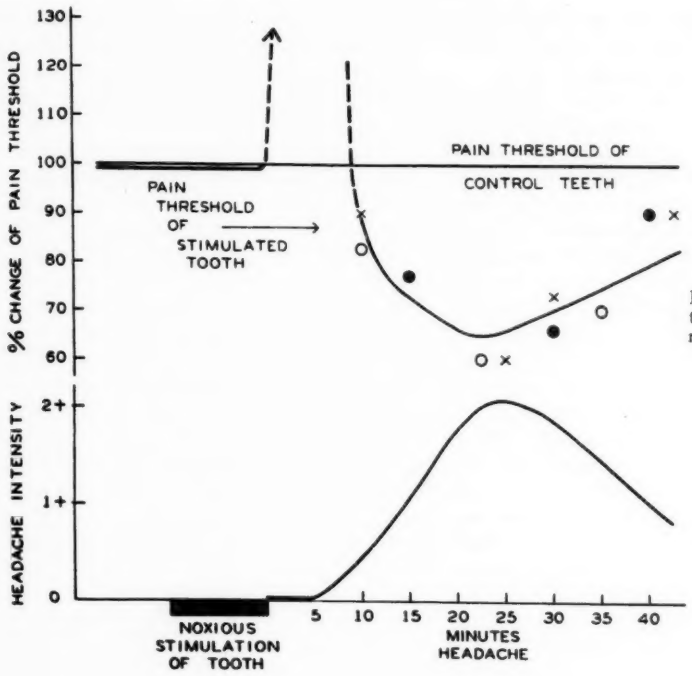

Fig. 40. Lowering of the pain threshold after noxious stimulation of a tooth.

tooth, the source of noxious stimulation. This explains why so many contradictory reports are made concerning the use of local analgesia. If the hyperalgesia be the greatest source of discomfort, effects of local analgesia may be striking; but if the pain from the source of noxious stimulation is the major feature, then local treatments of the hyperalgesic area are less effective in relieving discomfort.

Let us further discuss this topic of major interest to you as dentists—pain from the teeth. An attempt was made in my laboratory a few years ago with my colleagues Robertson and Goodell to ascertain the distribution and mechanism of headache and other pain in the face and head resulting from noxious impulses originating in the teeth. The effects of experimental noxious stimulation of the teeth by means of electrical stimulation, and of pain arising from morbid processes were studied. My associates used their own teeth for study and made careful observations of their sensations. Noxious stimulation of one or more of the upper teeth resulted in pain that was experienced at first locally and then diffusely in tissues supplied by the second division of the fifth cranial nerve. (Figure 38). Noxious stimulation of one or more of the lower teeth also caused pain to be experienced, at first locally and then widely, in the tissues supplied by the third division of the fifth cranial nerve. Figure 39). Often moreover, the site at which pain was felt was remote from the primary source of impulses set up at the site of injury. If the noxious stimulation of the teeth was sufficiently intense and prolonged, regardless of site of origin, the pain usually spread and was also experienced in tissues supplied by the first as well as by the second and third divisions of the fifth cranial nerve. The size of the painful area bore no close relation to the size or localization of this noxious stimulation, but pro-

longed disturbances were more likely to give rise to distant pain than were short-lived stimulation.

Furthermore, homolateral hyperalgesia, tenderness and vasomotor reactions of tissues remote from the source of impulses in the teeth, such as tender eyeball, reddening of the conjunctiva, and tenderness of the temporal and auricular tissues, were common .

During the period of twenty to thirty minutes after noxious stimulation of the tooth, when the intensity of the headache in the temporal area was at its maximum, the pain threshold of the stimulated tooth, now no longer aching itself, was lowered 35 per cent below its control level. (Figure 40). The pain thresholds of the adjacent control teeth were not altered. The experimental tooth was also painful when cold air was sucked through the mouth, or with drinking cold water. Two such experiments were performed on subject R., 2 on subject G.

These observations on lowering of the pain threshold in the teeth are also in keeping with the common experience that ordinarily nonnoxious stimuli, such as cold or hot water, in the mouth can induce pain in a tooth when it is diseased. Edema, dilation of blood vessels, and cardiac systole when there is inflammation and lowered pain threshold, also become adequate stimuli and give toothache its characteristic throbbing quality. Furthermore, it was found during high altitude flying that a diseased tooth that did not give rise to pain on the ground became painful with a drop in the barometric pressure. The sooner the pain starts during ascent, the more acute the inflammation in the tooth is likely to be, indicating a low pain threshold in the affected tooth.

In short, for a tooth with a lowered pain threshold, ordinarily inadepuate stimuli become noxious stimuli and give rise to pain. It is postulated that the experimental noxious stimulation of the tooth results in a local inflammatory reaction within the tooth, which outlasts the period of experimental stimulation, and perhaps also in the liberation of a substance which lowers pain threshold. Because of the lowered pain threshold minor tissue changes, such as vasodilatation and edema, become capable of evoking impulses which incite central spread of excitatory effects. These are ultimately experienced as headache remote from the site of stimulation.

On August 30 for therapeutic reasons subject R. had his third left upper molar extracted at 11:50 A.M. The tissues around the tooth were infiltrated with procaine hydrochloride before the extraction, which was quickly performed without pain. One hour and

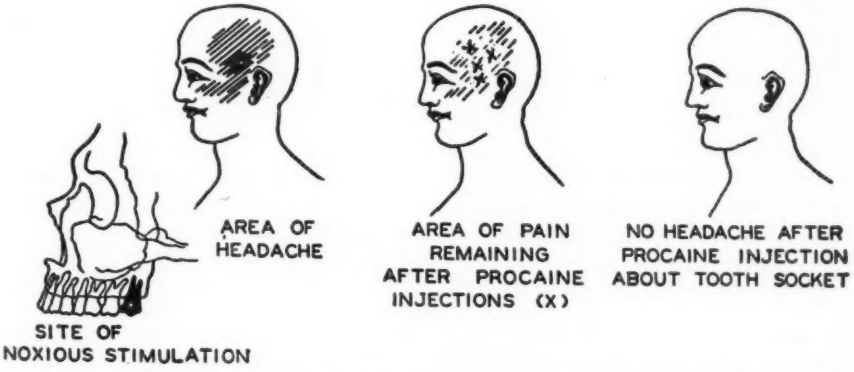
AREA OF HEADACHE
SITE OF NOXIOUS STIMULATION
AREA OF PAIN REMAINING AFTER PROCAINE INJECTIONS (X)
NO HEADACHE AFTER PROCAINE INJECTION ABOUT TOOTH SOCKET

Fig. 41. Site of noxious stimulation, area of headache, area of pain remaining after procaine injections, no headache after procaine injection about tooth socket.

twenty minutes after the extraction, however, the analgesic effect of the procaine was ended, and he experienced a slight pain and tenderness in the tooth socket. Ten minutes later there developed a headache similar in every respect to that following the experimentally induced toothache. However, it was of greater intensity and covered a greater area over the forehead and up to the vertex, beyond its zone of maximum intensity in the temporal area (Figure 41). There was lacrimation and slight reddening of the conjunctiva of the left eye, and erythema of the left side of the face, which extended below the jaw into the neck. The skin over the painful area, especially in the temporal area, was hyperalgesic, and pin prick in this area was accompanied by an extensive pilomotor reflex as evidenced by cutis anserina over the left side of the neck, shoulder and arm.

At 1:50 P.M. the headache was moderately intense and there was a slight pain in the tooth socket. One cc. of procaine hydrochloride (1 per cent) was injected intracutaneously into the temporal region at the site of maximum intensity of pain, and over the area of deep tenderness. The patient reported that the 'center' of his headache was eliminated, leaving a fringe of slight to moderate headache and a deep diffuse ache beneath the area of surface analgesia. The slight pain in the tooth socket remained unchanged.

Again at 2:20 P.M. two additional areas were injected intracutaneously with procaine, and at 2:30 since deep ache still remained, two intramuscular injections were made fanwise into the temporal muscle, as indicated in Figure 41. The subject continued to experience pain of 2 plus intensity at the periphery of the analgesic area but he also described a diffuse ache of low intensity which seemed to stem from the analgesic area.

At 4:00 P.M. the effects of the injected procaine had diminished, and the headache was again of moderate intensity. The subject reported: "This is the same headache I had following experimentally induced toothache, except that it is more persistent, and of slightly greater intensity. The tooth socket is painful." At 4:10 P.M. the left middle superior alveolar nerve was blocked with monocaine. All of the headache, including the sensations of fullness and tightness, was eliminated at once, and the subject experienced no discomfort until about three hours later when the action of the analgesic was dissipated.

Another pertinent observation was made in a 38 year old nurse who had an 'excruciating'

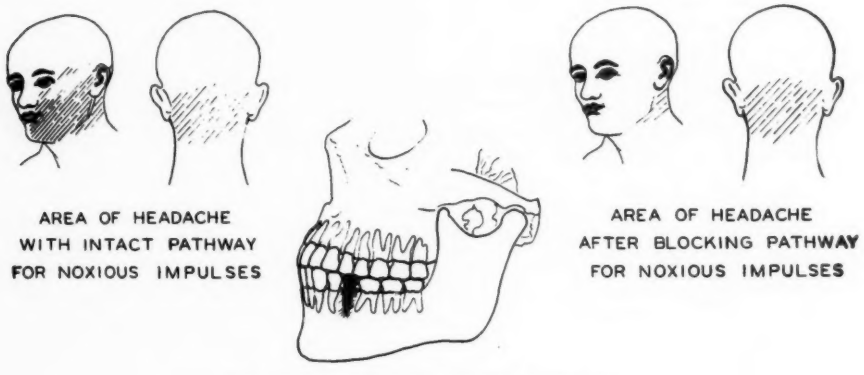

Fig. 42. Site of origin of noxious impulses (osteomylitis); area of headache with intact pathway for noxious impulses; area of headache after blocking pathway for noxious impulses.

toothache and headache of high intensity of two weeks' duration associated with osteomyelitis of the right lower jaw. The headache extended from the midline of the chin along the lower jaw to the top of the right ear and into the ear, into the upper jaw, into the neck below the jaw, and across the back of the head. All of the lower teeth on the right ached, and were tender on slight pressure. There was intense pain at the angle of the jaw, and the jaw could be opened no more than ½ cm. because of the contraction and extreme tenderness of the masseter muscle. A monocaine block of the mandibular nerve instantly eliminated all of the pain in the face. The teeth remained tender to pressure for three to four minutes until analgesia of the entire lower right side of the jaw was complete. Then the mouth could be opened with but slight discomfort. There remained tenderness and pain in the right side of the neck, and both superficial and deep tenderness and headache over both sides of the back of the head and neck (Figure 42).

These observations demonstrate further that the pain resulting from central spread of excitation over all sensory portions of the involved segments is immediately abolished when the primary source of noxious impulses is blocked.

The speed of elimination of the headache after blocking of the path of afferent impulses from the injured periodontal tissues of the tooth socket makes it likely that the diffuse headache experienced was caused by the afferent impulses arising from these injured tissues. These afferent impulses gave rise to excitatory processes in the brain stem which spread to exert their effects on many trigeminal structures.

In addition, however, these observations show that sustained conraction of the muscles of the head and neck secondary to impulses from tissue damage arising in any part of the head become in themselves a basis of complaint and headache. These persist for some time after elimination of the primary source of impulses from adjacent tissue.

Such residual muscle pain is similar to that which occurs after the vascular components of migraine headache have been eliminated by ergotamine tartrate. In the case of the teeth, this pain from sustained muscle contraction is notably in the masseter and temporalis muscles, in addition to the muscles of the occiput and neck. Occasionally, therefore, pain from such sustained muscle contraction may be a dominant feature of the discomfort from afferent impulses arising in the teeth.

Afferent impulses arising from the teeth and associated with headache, although clinically important, are not a common cause of headache. Headache due to a diseased tooth without toothache or local tenderness near the tooth is rare. The teeth as a cause of 'neuralgia' or other face pain do not remain obsure. Despite the suggestion that tooth inflammation, impaction and exposed dentine may be hidden causes of headache and other pain, clinical experience makes it appear likely that if such conditions are the sources of noxious impulses they make themselves obvious to the patient through spontaneously occurring local pain or through pain induced by thermal, mechanical, or chemical stimuli. Furthermore, it is possible to ascertain whether a given diseased tooth or teeth are the sources of impulses relevant to pain and the cause of headache by answering the following question: Does injection of monocaine into the tissues about the tooth or teeth in question eliminate the headache and other pain, leaving only tenderness and ache in the muscles of the neck and back of the head? Unless the face or head pain is dramatically reduced or eliminated by such measures, such a tooth, even though seriously diseased, is not the cause of the headache or 'neuralgia'.

Certainly no recurrent or periodic headache attacks, or pain in the face or head of years' standing, with intervals of complete freedom from pain in the interims between attacks, can have their origin in morbid dental processes. Migraine, atypical 'neuralgia', or major trigeminal neuralgia never stem directly from dental disease.

One cannot end a talk on headache without mentioning allergy. All I can say is, that if an allergic reaction involves the nasal mucous membranes, by virtue of dust, pollen or gasses,

it may give rise to painful sensations, but an allergic reaction to ingested foods probably has little to do with headache. I then would draw this long discussion to a conclusion by asking you to consider the headache problem in its broadest terms. More than 90 percent of people with headache, have their headache as a result of vascular disturbances and sustained skeletal muscle contraction about the head, and vasomotor disturbances within the nose. A small percent have their headache as a result of serious intracranial growths, hemorrhages, infection or irreversible alterations in structure and only a very small percent have head pains because of defects in their teeth.

Often the simplest and most effective therapeutic agent may be the doctor's sympathetic relationship with the patient, and his kindly, helpful attitude. Necessary may be a more penetrating attempt to have the individual with headaches recognize and understand those factors in his life that may be causing him to go in the wrong direction, or at the wrong pace. Two people out of three, with these vascular muscle tension headaches can be greatly helped by any physician who is interested in human problems, and who is willing to spend a minimum of time with his patients in reviewing them. Some will prefer to keep their way of life and worry along with their problems . . . willing to put up with headaches when they are assured they are not ominous symptoms. But unfortunately, there will be the occasional patient who is caught in a morass of hates and hostilities, futilities and hopelessness, that nothing can change. He will go from doctor to doctor, from clinic to clinic, and often enough to dentists, too, looking for an operation, a tooth extraction, a diet, a pill that will cure him. After a long and miserable life he continues to have his headaches, and frustrates those who try to help him.

In concluding therefore, I would urge you to take a positive and constructive attitude toward headache in individuals who may have concurrent eye, nose or tooth defects, indicating that in some this may be a factor. But please remember the basic personal and interpersonal difficulties that are often far more relevant. You as interested humans can help to alleviate much suffering.

BIBLIOGRAPHY

1. Wolff, H. G.: *Headache and Other Head Pain* Oxford University Press, New York, 1948.
2. Robertson, Schuyler; Goodell, Helen; and Wolff, H. G.: Studies on Headache: the teeth as a source of headache and other pain. Arch. Neurol. and Psychiat. 55:277, 1947.
3. Tunis, M. Martin; and Wolff, H. G.: Studies on Headache: Cranial Artery Vasoconstriction and Muscle Contraction Headache. Arch. Neurol. and Psychiat. 71:425, 1954.
4. Goodell, H.; Lewontin, Richard; and Wolff, H. G.: The Familial Occurrence of Migraine Headache: A Study of Heredity Proc. of Assoc. for Res. in Nerv. and Ment. Dis. Vol. XXXIII Williams and Wilkins Co., Baltimore, 1954, p. 346.
5. Ostfeld, Adrian M.; Reis, Donald J.; Goodell, H.; and Wolff, H. G.: Headache and Hydration: the significance of two varieties of fluid accumulation in patients with vascular headache of the migraine type Arch. Int. Med. 96:142, 1955.
6. Wolff, H. G.: Headache Mechanisms Intern. Arch. Allerg. and Aplied Immunol. Vol. 7, No. 4-6, 1955.
7. Pichler, Ernst; Ostfeld, A. M.; Goodell, H.; and Wolff, H. G.; Studies on Headache: Central versus Peripheral Action of Ergotamine Tartrate and its Relevance to the Therapy of Migraine Headache. Arch. Neurol. and Psychiat. 76:571, 1956.
8. Ostfeld, A. M.; and Wolff, H. G.: Studies on Headache: Participation of Ocular Structures in the Migraine Syndrome. Modern Problems in Ophthalmology Vol. 1 New York S. Karger, 1956.
9. Ostfeld, A. M.; Chapman, Loring F.; Goodell, H.; and Wolff, H. G.: Studies in Headache: Summary of Evidence Concerning a Noxious Agent Active Locally During Migraine Headache. Psychosomatic Med. Vol. XIX:3, 1957.

Appreciation is expressed to Karger and Co., publishers of the International Archives of Allergy and Applied Immunology and to the Sandoz Chemical Company, for making possible the use of the engravings of many of the illustrations in this article.

THE DENTIST AND VOCATIONAL REHABILITATION

HOWARD A. RUSK, M.D.*

and

SAMUEL S. HERMAN, D.D.S., Ph.D.†

Rehabilitation is frequently termed the "third phase of medicine," following preventive medicine and curative medicine and surgery. It is that period in patient care when "the fever is down and the stitches are out," the period "between the bed and the job." In contrast to convalescence in which the patient is left alone to let nature and time heal and restore, rehabilitation denotes a dynamic program aimed at the ultimate restoration of the disabled person to his maximum capacity—physical, emotional, social, and vocational. Implicit in the concept of rehabilitation is the need for a multi-disciplinary team whose efforts can be integrated as a single force to assist the disabled person in attaining his maximum potentials. Needless to say, no one profession can furnish all the services needed, nor has an independent specialty of rehabilitation developed. Rehabilitation is, rather, an area of specialized activity within a number of professional disciplines.

Dental care is frequently an essential element in rehabilitation of the handicapped. For maximum effectiveness, however, it must be fully coordinated with the services of physicians, physical therapists, occupational therapists, nurses, social workers, vocational counselors, and other trained personnel. The State-Federal program of vocational rehabilitation is an important mechanism for effecting such coordination. Moreover, it offers to the dental practitioner an opportunity to enlarge his role in a Nationwide attack upon prolonged disability.

The State-Federal program of vocational rehabilitation is characterized by its primary concern for those disabled persons, who can become economically independent. Its objective is to help physically and mentally impaired persons, through the provision of substantial rehabilitation services, to be placed in gainful employment, including sheltered workshop or home employment. These services are extended also to persons who can contribute to the national economy in other ways, as for example, to homemakers who will be enabled thereby to perform homemaking duties.

ORGANIZATION AND ADMINISTRATION

The program is organized and administered as a State-Federal partnership. Federal responsibilities are discharged by the Office of Vocational Rehabilitation in the Department of Health, Education, and Welfare. In addition to its responsibility to exercise national leadership to the program, the Office provides a comprehensive program of assistance to the States for vocational rehabilitation. Federal matching grants are made to States for providing rehabilitation services to the disabled and for encouraging them to offer new services and to cover new geographic areas. The disbursement of Federal grants-in-aid, however, is only one phase of the cooperative program with the States. The Office also makes studies, conducts investigations, and disseminates information on the needs and capabilities of the handicapped. It provides consultation on specialized aspects of rehabilitation and assists the States in the interpretation of their programs to the public. Most of these technical and consultative services reach State agencies through the nine regional representatives of the Office and their staffs.

Actual services to the disabled are provided by public agencies in all of the States and territories. Thirty-five States and Hawaii have one agency serving the blind and a separate agency

* Chairman of the Department of Physical Medicine and Rehabilitation at the New York University—Bellevue Medical Center.

† Chief, Division of Medical Services and Facilities, Office of Vocational Rehabilitation, Department of Health, Education, and Welfare.

concerned with rehabilitation of persons with all other types of disability. In the remaining States, all disability groups come within the purview of a single agency. Each agency operates its own program in accordance with a State plan developed in cooperation with the Office of Vocational Rehabilitation. These plans describe the organization of the program, standards of personnel administration, and the policies governing program operations. To insure flexibility, State plans may provide for a decentralization of administration to county or municipal agencies.

ELIGIRILITY AND SERVICES

The process of rehabilitation begins with case finding. Disabled persons are referred to a State rehabilitation agency from many sources—private practitioners, hospitals, health departments. A major case-finding device is the cooperative agreement between State vocational rehabilitation agencies and such organizations as employment services, public assistance agencies, and crippled children's divisions. For the future, two recent amendments to the Social Security Act are likely to assume importance as sources of referral. The first, enacted by the 83rd Congress, is the so-called "disability freeze" which preserves the insurance rights of eligible individuals who become totally disabled while covered by Old-Age and Survivors Insurance. The second is the authorization in 1956 for cash payments to covered workers aged 50 and over who become disabled and unable to perform substantial work. In so doing, Congress also stressed the importance of rehabilitating these disabled individuals in every possible case, and called for the referral of cases to the State vocational rehabilitation agency. Provisions were also made for the determinations of disability to be made by a State agency, acting for the Old-Age and Survivors Insurance System, with the State rehabilitation agencies indicated as the agency of choice.

Generally, the criteria for eligibility are these: an individual must have a physical or mental disability which is a substantial handicap to employment; he must be of, or near, working age; and there must be a reasonable expectation that the service will render him fit to engage in remunerative employment. The actual determination of eligibility in each case, however, is a responsibility solely of the State agency.

The client receives a thorough diagnostic study in which all factors influencing the course and outcome of rehabilitation are evaluated. To the degree needed, this study embraces the individual's personality, intelligence, educational status, work experience, vocational aptitudes, personal and social adjustment, and employment opportunities. On the basis of these data an individual plan of vocational rehabilitation is formulated, which sets forth a vocational objective and the services necessary to attain this objective.

Comprehensive services are available to the disabled under this program, These include medical diagnostic services, individual counseling and guidance, physical restoration, training for a job, transportation, maintenance, occupational tools, initial stocks of goods for small business enterprises, job placement, and follow-up. Medical diagnosis, counseling, placement, follow-up, and in most cases training are furnished without cost. Public funds are used in providing all other services to the extent that the disabled person is not able to pay for them.

PHYSICAL RESTORATION

In its early years, vocational rehabilitation consisted largely of an attempt to "get around" the disability by specialized job training and by helping the disabled to find work. Action to eliminate or reduce a handicap through medical services was first authorized by Federal legislation in 1943. Increasing emphasis in the medical direction since that time is reflected in the fact that physical restoration services are received by more than half of those now being rehabilitated. Moreover, they currently account for more than 40 percent of the expenditures for case services to clients.

Included in the term "physical restoration" are such services as medical and surgical treatment by general practitioners or specialists, psychiatric treatment, dentistry, nursing, hospitali-

zation and clinic services, convalescent or nursing home care, drugs, prosthetic devices, physical therapy, occupational therapy, and medically directed speech or hearing therapy. These services are made available only in instances where (1) the clinical status of the individual's condition is stable or slowly progressive (i.e., not acute or transitory); (2) where a favorable outcome may be expected within a reasonable period of time; and, (3) where the individual's financial need has been demonstrated.

State agencies do not duplicate facilities, personnel, and equipment that are already available elsewhere. Services to the client are purchased from physicians, dentists, hospitals, and other facilities in the community. With respect to rates of payment, the Office of Vocational Rehabilitation looks to the State agencies to establish their own administrative controls to assure efficient administration and high quality of service. The Office consistently recommends that professional advisory committees and appropriate professional organizations in the State be utilized in determining rates of payment.

DENTAL SERVICES

In establishing standards and criteria governing the provision of dental services, the Office of Vocational Rehabilitation has worked closely with the dental profession. For example, the American Dental Association's Council on Dental Health has cooperated with the Office in the development of policies and guidelines for the use of State agencies. These guidelines help to identify circumstances in which dental defects may be considered vocationally handicapping, and hence, as indications for dental care as a part of physical restoration.

Foremost among these indications are the disabilities observed in persons with cleft palate, in those who have undergone extensive facial surgery for removal of a malignancy, and in those who have sustained injuries to the face, teeth, and jaws as a result of automobile accidents. In these instances, unintelligible speech, cosmetic deformity, and inability to eat may represent substantial employment handicaps. To reduce these handicaps, the dentist is called upon to provide services ranging from general operative procedures to complex prostheses and appliances.

Although dental defects may not in themselves result in inability to perform specific occupations, they may directly contribute to the severity of other conditions which are job handicaps. Thus, for example, dental service may be a primary component in the physical restoration of individuals with severe peripheral facial paralysis traceable, in the opinion of examining neurologist and dentist, to a dental focus of infection.

Another, perhaps more frequent, indication for dental service in the vocational rehabilitation program arises in connection with an employment handicap which stems from multiple disabilities. Where a combination of disabilities makes it impossible for the individual to work, it may be necessary to furnish every corrective measure, including comprehensive dental care, to bring about a state of health conducive to satisfactory functioning on the job.

The provision of dental care to persons with multiple disabilities is probably found most frequently in the case of cerebral palsy. This results from the recognition given by The National Society for Crippled Children and Adults and, particularly the United Cerebral Palsy Associations, to the dental problems of children and young adults affected by cerebral palsy. Since 1950, the United Cerebral Palsy Associations, for example, have sponsored a number of fellowships at the Columbia University School of Dental and Oral Surgery under which program two Fellows in Dentistry have been appointed annually for a 2-year period to gain proficiency in providing dental care for the cerebral palsied person.

A particularly interesting film of the dental problems of children with cerebral palsy and other disabilities is "Dentistry for the Handicapped Child" produced as a professional and public service by the A. H. Robbins Company, Richmond, Virginia, under the technical direction of Dr. Manual M. Album, University of Pennsylvania.

A rather unique program involving dentistry for those with multiple disabilities is that which has been developed by Abilities, Inc., the highly successful manufacturing plant in

Long Island which employs only severely disabled persons. Abilities, Inc., started in the summer of 1952 with four employees in a vacant garage. Two of these men were in wheelchairs and among the four employees were but one usable leg and five usable arms. No charity was accepted, the original capital was borrowed and repaid with interest. The firm now occupies a modern $750,000 building of 45,000 square feet and employs over 350 severely disabled persons. Early in its operation, Abilities, Inc.'s founder and president, Henry Viscardi, Jr., himself legless, was struck by the dental neglect among his increasing number of employees.

This dental neglect resulted not only from the fact that most of his employees had never worked before or had not worked for long periods, but that most dentists in Nassau County have steps leading to their offices or their offices are on the second floor of buildings without elevators. Viscardi's employees — paraplegics, amputees, and cerebral palsied — in wheelchairs and on crutches could not get up the stairs to their offices.

Viscardi contacted the Nassau County Dental Society which appointed a committee to work with him on the problem. The result is a modern, completely equipped dental unit in the new building. All new employees are provided a dental survey. Services are then provided on a fee-for-service basis by dentists appointed by the dental society. At first, the Nassau County Dental Society offered to provide dental services without charge, but Viscardi and his workers refused. They are proud that despite their severe disabilities, they are employed, tax-paying citizens who accept and want to meet their own responsibilities.

Since dental services are clearly within the scope and purpose of the vocational rehabilitation program, the Office recommends that State agencies establish no restrictions as to type of service nor maximum amounts that may be expended per case. While any type of dental treatment needed to remove the handicapping effects of a dental or associated disability has a place in physical restoration, it is not a function of the vocational rehabilitation program to provide general dental care. In common with all other medical and medically-related services, dental treatment is authorized only where it will have a direct and demonstrable effect upon a client's employability.

THE BROADER FRAMEWORK

During the fiscal year ending June 30, 1958 a record total of 74,320 handicapped persons were placed in remunerative employment suited to their capacities. This was 3,380 more than the 1957 record of 71,607. It is estimated that these men and women rehabilitated through the public program last year will increase their annual earning from $20.7 million to about $141.1 million in their first full year of employment and will add 109,000,000 man-hours to the nation's productive effort.

In 1958, 18,584 persons successfully completed rehabilitation services but had not yet found employment due to the economic recession. Estimates indicate that yearly earnings of the 74,320 men and women rehabilitated through the public program will increase from $22,000,000 to $148,000,000 in their first year of employment.

Although this represents a considerable achievement, there is still much to be accomplished. Today, at least two million Americans, disabled because of disease, accident, or congenital conditions, could be helped by vocational rehabilitation. This backlog of neglected disability, maintained by an annual increment of 250,000 persons who could profit from these services, reflects persistent deficiencies in funds, facilities, manpower, and research. The Vocational Rehabilitation Amendments of 1954 —the first comprehensive legislation in this field since 1943—provided the basis for a concerted effort to meet the deficits.

First, the new law provided for an appreciable increase in grant-in-aid funds for the State agencies, and for an incerased flexibility in the use of this money. It also made Federal funds available for the first time to voluntary groups which can contribute to the Nationwide expansion of services.

Another provision of the law permits the use of Federal funds for remodelling or altering existing buildings to render them suitable for

use as facilities for rehabilitation of the severely disabled or for sheltered workshops. Also, Federal funds may be used to procure initial equipment and to pay for staff during the first year of operations. This feature of the law is closely articulated with provisions for construction of rehabilitation facilities under the Medical Facilities Survey and Construction Act of 1954.

Among the funds authorized are those for training rehabilitation personnel. Grants are now awarded to educational institutions for the establishment and improvement of professional curricula in the various disciplines involved in rehabilitation. Grants are also made to assist qualified students in meeting the costs of professional training in the shortage fields, as well as of advanced training in preparation for teaching careers. Further, the Office of Vocational Rehabilitation helps to underwrite the costs of short courses outside the regular curriculum, as well as the costs incurred by those attending the courses.

Finally, the 1954 amendments make it possible to move ahead in the area of fact-finding, applied studies, and demonstration. Such projects are critically needed if the benefits of basic and clinical research are to be brought to those who have the responsibility for furnishing rehabilitation services. Upon recommendation of a National advisory body, the Office awards grants to public and private nonprofit organizations for the conduct of special projects designed to solve vocational rehabilitation problems of national significance.

The enactment of this law has brought vocational rehabilitation to the threshold of a new and more effective era of operation. Its basic principle is that of channeling the fullest benefits of modern rehabilitation know-how to the disabled client. Application of the principle demands the closest type of teamwork among the disciplines and agencies interested in the problems of the handicapped. In this cooperative effort the dentist will find both opportunity and challenge.

The growing interest throughout the world, both professionally and among the public, in the development of rehabilitation services for the physically handicapped is one of the most significant social advances of recent years. Originally, much of this interest in expanding rehabilitation opportunities and services for the handicapped resulted from World War I, when attention was focused on the problems of disabled servicemen. In the past decade, the growth of rehabilitation services for the handicapped has been a part of an expanding community, national and global emphasis on social welfare, which is manifest in comparable advances in other educational, health and social services.

The heightened interest in rehabilitation has been primarily a result of the impact and recognition of increased needs. Although many nations of the world still have pressing problems of communicable disease, and life expectancy rates vary from nation to nation, the trend in most nations is toward a long life expectancy. The rapid increase in the more industrialized parts of the world of the incidence of chronic disability follows from the increasing age of the population.

Moreover, as the average age advances in the more developed parts of the world, a new concept of the dignity of man and the value of the worth of the individual is beginning to emerge in those nations of the world which do not yet benefit from modern programs of public health and medical care. This new concept of the worth of the individual, long a part of life in industrial countries, is symbolized in many instances in the desire of developing nations to institute programs of services for their physically handicapped.

New York University-Bellevue Medical Center, New York, N. Y.

LE DENTISTA E LE REHABILITATION VOCATIONAL

Dr. med. Howard A. Rusk e Dr. chir. dent. e phil. Samuel S. Herman

SUMMARIO IN INTERLINGUA

Le rehabilitation es frequentemente nominate "le tertie phase del medicina," post le phases del medicina preventive e del medicina e chirurgia curative. Illo es le periodo in le tractamento del patiente quando "le febre es abattite e le filos es removite," le periodo "inter lecto e travalio." In contrasto con le convalescentia, quando le patiente es lassate in pace pro permitter al natura e al tempore de resanar e restaurar le, le rehabilitation denota un programma dynamic que visa al ultime restauration del invalidate persona a su maxime capacitate physic, emotional, social, e professional. Implicite in le concepto de rehabilitation es le necessitate de un equipa multi-disciplinari cuje effortios pote esser integrate como un fortia unic pro assister le persona invalidate de attinger le maximo de su potentialitates. Il va sin dicer que mulle profession individual pote provider omne le servicios necessari, e un independente specialitate de rehabilitation non se ha disveloppate. Rehabilitation es plus tosto un campo de activate specialisate intra un numero de disciplinas professional.

Tractamento dental es frequentemente un elemento essential in le rehabilitation de personas incapacitate. Pro le maxime efficacia, nonobstante, le tractamento dental debe esser coordinate con le servicios de medicos, physiotherapistas, therapistas occupational, infirmeras, agentes de prevision social, consilieros professional, e altere specialistas. Le programma Statal-Federal de rehabilitation professional es un mechanismo importante pro effectuar un tal coordination. In plus, illo offere al practico dental le opportunitate de amplificar su rolo in un attacco general in omne partes del pais super le problema del incapacitation prolongate.

CONGENITAL DENTAL DEFECTS*

SEYMOUR J. KRESHOVER, D.D.S., M.D., Ph.D.**

Developmental defects of the teeth have long been the subject of clinical and experimental study. However, in almost all instances the etiologic relationship of various systemic disorders to these abnormalities has been established with reference to the postnatal period. It is not surprising, therefore, to note the limited information available on the subject of prenatal influences on tooth formation. Among the few reports to be found in the literature are those of Stein, who reported 8 cases of enamel hypoplasia involving the incisal third of deciduous teeth[1]; Sheldon, Bibby and Bales, who noted hypoplastic defects of intrauterine origin in 5 out of 34 individuals studied[2]; and Wolfe[3] and Diamond and Weinmann,[4] who respectively called attention to the significance of such maternal influences on dental development as rickets and syphilis. In experimental studies, Mellanby noted abnormally shaped incisors and molars in the offspring of rats that had been maintained on vitamin A deficient diets prior to breeding.[5]

In addition to the importance of gaining more information on etiologic factors in developmental dental defects, the fact that teeth can show a pictorial record of disturbances suggests their value as a tool for investigation of congenital abnormalities other than dental. In other words, any disturbance in the highly specialized function of odontogenesis that is manifested in teeth that had undergone development prior to birth might be indicative of a more widespread fetal response to adverse maternal influences. This type of information might also aid in the further evaluation of the placenta as a barrier to, or pathway for, transfer of certain injurious agents from mother to fetus.

MATERIALS AND PROCEDURE

A. Human

In order to explore some of the many questions related to prenatal influences on dental development, a selected series of human subjects was studied.[6] Of the 35 infants examined (ages ranging from premature stillborns to two and one-half months), 27 showed disturbances of tooth formation and 8 were negative. It should be emphasized that the total series of 35 included various deformities (dental and non-dental), as well as various causes of interruption of pregnancy. On the basis of information provided by length of gestation, duration of life and measurement of enamel and dentin apposition, it was possible to date the abnormal cases into 7 of prenatal onset, 15 that occurred during the neonatal period, 4 that were of postnatal occurrence, and one that was related to both the prenatal and postnatal period (Figures 1, 2, and 3). Arbitrarily establishing the neonatal period as including the first 14 days after parturition, all 15 abnormalities dated to this period were of such severity as to be readily differentiated from physiologically occurring neonatal lines.

Based on pediatric histories and autopsy findings, 9 of the 35 babies studied showed multiple congenital anomalies such as spina bifida, hydrocephalus, abnormal rotation and position of the alimentary tract, polydactylism, and clubbed feet. In 7 of these cases, dental defects were also present whereas 2 of the babies showed no dental abnormalities. Of some interest was that, with the exception of 3 mothers presenting respective histories of anemia, preeclampsia and vaginal bleeding, the courses of pregnancy in these 9 cases were seemingly uneventful. Apparently, no clear-cut etiologic picture existed to account for either the dental defects or the other anomalies observed. However, a genetic origin must always be a consideration.

*Presented at the 14th Annual Meeting of The American Institute of Dental Medicine, Palm Springs, California, October 13-17, 1957.

**Associate Director, National Institute of Dental Research, National Institutes of Health, Public Health Service, U. S. Department of Health, Education, and Welfare, Bethesda, Maryland.

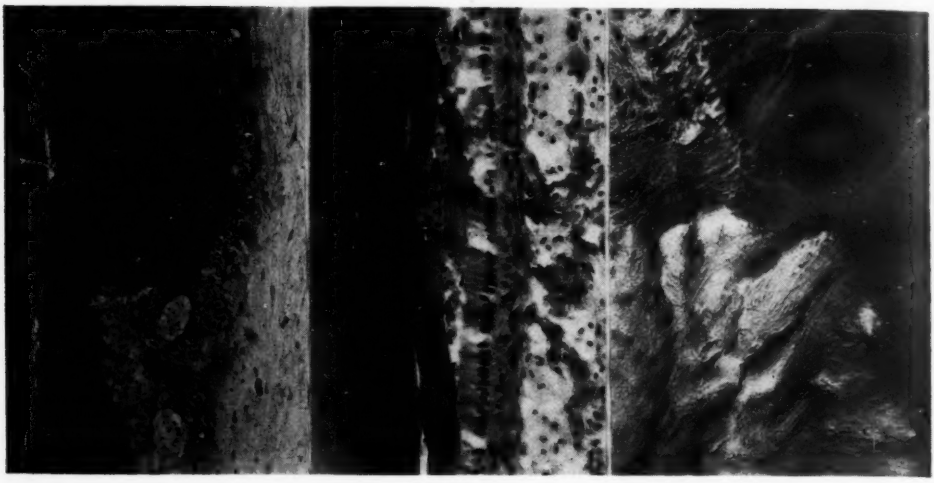

Fig. 1 Fig. 2 Fig. 3

Fig. 1.—Lower left canine of infant that lived 18 hours. Noted at autopsy were multiple congenital anomalies including extrophy of bladder, imperforate anus, and atresia of colon. Duration of pregnancy was 8½ months, with an uneventful course except for hypochromic normocytic anemia. Note the ameloblastic degeneration in the area of aplastic enamel. This abnormality is dated to the prenatal period.

Fig. 2. Neonatally occurring abnormality in the lower left incisor of a 14 day old baby. There is some disorganization of the ameloblastic layer and formation of an amorphous, enamel-like material overlying the organic enamel matrix. Scattered pyknotic nuclei of degenerating ameloblasts are noted in the area. Autopsy examination showed an aspiration pneumonia. Pregnancy was uneventful with spontaneous, premature delivery at 6½ months.

Fig. 3.—Enamel defect in lower right molar of an 11-week old baby delivered spontaneously after 8 months of essentially uncomplicated pregnancy. Anatomical diagnosis included congenital heart disease, gargolyism, and pulmonary congestion and edema. Based on micrometer measurements of enamel matrix formation, it is possible to date the dental defect to the postnatal period.

Seven other babies, in the series of 35 studied, developed meningitis in the immediate postnatal period. Three of these presented dental defects of apparently postnatal onset that could be correlated with the meningitic infection. Interestingly enough, one of these infants also showed a prenatally dated lesion that could be correlated with a maternal syphilitic infection. The mother of a fourth baby with prenatally formed dental abnormalities had a similar history of syphilis. In this instance, however, there were no postnatal dental changes that could be correlated with the meningitis. The remaining 3 infants in the series of 7 with meningitic infections failed to show evidence of any dental abnormalities.

Another group of 15 infants (in the total series of 35) had a history of pneumonia and pulmonary atelectasis. Of this number, 12 had dental defects of neonatal or early postnatal onset that could be correlated with the pulmonary lesions. The remaining 3 babies showed no dental deformities.

Of the remaining 5 infants in the total series of 35 studied, 4 showed abnormal odontogenesis of prenatal onset. In all cases, the maternal histories were negative.

B. Experimental

As indicated by the reports of many previous investigators,[7, 8, 9] a wide variety of metabolic disturbances can cause abnormalities in teeth that are undergoing active formation at

the time of systemic injury. However, it is not clearly established whether metabolic disorders in the gestational maternal organism can similarly affect the oral structures of the young in utero. In order to study this question, a series of experiments were undertaken in pregnant rats.

1. Pyrexia.

Since fever is a common accompaniment of many disease states, it seemed that it, in itself, might exert a significant influence on the developing oral structures. Although the literature contains references to dental changes in animals rendered pyrexic,[10, 11] practically no attention has been given to an evaluation of the response of fetal dental tissues to elevated temperatures in the gestational mother. In order to study this question, 28 pregnant rats were subjected to artificially-induced fever by means of an incubator equipped with ventilation and humidity controls.[12] In accordance with their stage of pregnancy, as determined by the presence of spermatozoa in vaginal smears, the rats were divided into 3 groups, each rendered pyrexic during a different trimester of pregnancy. By adjustment of incubator controls, the rectal temperatures of all animals were maintained in the range of 102 to 105 degrees Fahrenheit for periods of 12 to 48 hours. (Normal temperatures range from 98.5-99.5). Of the 28 rats so treated, 13 died during their stay in the incubator and 15 completed a normal course of pregnancy with spontaneous delivery at term. Sacrifice of litters was scheduled so as to provide jaw specimens of animals ranging in age from one to 60 days. After the weaning period, the mothers likewise were sacrified for study. Dental tissues of animals that died during their episodes of fever were also prepared for histologic examination. The finding of abnormal enamel formation in the pyrexic adult animals (Figure 4) was of particular interest in that previous reports in the literature called attention to changes in dentin formation but not in amelogenesis. With

Fig. 4

Fig. 5A

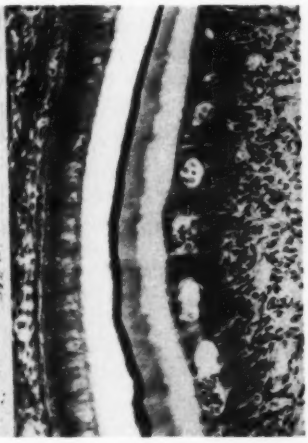

Fig. 5B

Fig. 4.—Developing incisor of rat with artificially induced fever of 29 hours duration. The ameloblastic zone shows large areas of cystic degeneration and ectopic formations of enamel-like material.

Fig. 5A.—Maxillary incisor of a 3-week old rat delivered of a mother that had been subjected to fever during the 3rd trimester of pregnacy. Note the defective matrix formation.

Fig. 5B.—Incisor of another offspring of a rat that had been rendered pyrexic during pregnancy. Particularly evident is the engorgement of large capillary vessels in the odontoblastic zone causing compression of adjacent cells.

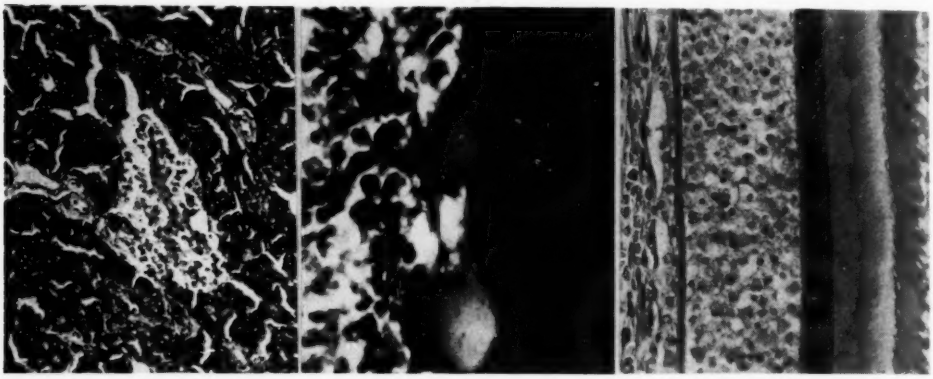

Fig. 6 Fig. 7A Fig. 7B

Fig. 6.—Islet of Langerhans in alloxan injected rat.

Fig. 7A.—Incisor of a rat injected with alloxan during the 3rd trimester of pregnancy. There is degeneration of the ameloblastic zone and deposition of two masses of enamel-like material on the surface of the organic enamel matrix.

Fig. 7B.—Incisor of a 12-day young rat injected with alloxan during the 3rd trimester of pregnancy. The ameloblastic zone shows a marked degenerative change.

reference to the offspring of these animals, abnormalities of both enamel and dentin formation were found in the incisors as well as in the molars (Figure 5). With but 2 exceptions, the mothers had been rendered pyrexic after the ninth day of gestation.

2. Alloxan diabetes.

Diabetes mellitus, as a metabolic disease, has been shown to have a distinct influence on the course of pregnancy and on the general development of the fetus.[13] However, although references to its influence on children's teeth have appeared in the literature[14, 15], seemingly little attention has been given to its effect on the formative dental tissues of the young in-utero. In order to pursue this question, the effect of alloxan on the course of pregnancy was studied in a group of 39 rats[16]. Intraperitoneal injections of the drug (155 mg. per kilogram of body weight) were made at varying periods after mating. Then, as deemed necessary, protamine zinc insulin was given and blood and urine sugar determinations made.

Although some animals failed to demonstrate a consistent hyperglycemic response, most showed a characteristic and persistent elevation of sugar levels, as well as a definite histologic picture of pancreatic islet damage (Figure 6). Observing the course of pregnancy, dated from the appearance of spermatozoa in vaginal smears, it was noted that the later in gestation that alloxan was given, the greater was the chance of a successful termination of pregnancy by spontaneous delivery. A similar direct correlation existed between time of injection and numbers of viable young in delivered litters; i.e., the later the injection, the greater was the number of living offspring. Of additional interest was that none of the newborn rats showed excessive weight or elevated blood sugars. With reference to findings in dental tissues, marked abnormalities were manifested in both the injected maternal animals and their offspring (Figure 7). These changes were characterized principally by disturbed amelogenesis, odontoblastic degeneration, and pulpal hyperemia and hemorrhage. The nature and degree of these changes had no correlation with the time of pregnancy when alloxan was given, or with the severity of hyperglycemia.

3. Virus Infections: vaccinia and lymphocytic choriomeningitis.

With reference to maternal infections as etiologic factors in congenital dental abnormalities, the role of the placenta assumes considerable importance. Studies evaluating the placenta as a barrier to, or pathway for, agents that might be causally related to fetal defects, have however, been necessarily conjectural. For example, although it has been suggested that such virus diseases as rubella, rubeola, mumps, and varicella are possibly of etiologic significance, the mechanism by which they may initiate congenital abnormalities has not been established. Certainly, consideration must be given to the significance of metabolic by-products of the maternal disease as well as to actual transplacental infection. In clinical studies of the effect of various maternal virus diseases on fetal dental development, a definite etiologic relationship has apparently been established with respect to rubella.[17] An important aspect of such studies is that disturbances in the highly specialized function of odontogenesis are probably indicative of a more widespread fetal response to adverse maternal influences, whether they be virus infection *per se* or some related metabolic disturbance.

The selection of vaccinia for study[18] was based on such criteria as (1) its ability to produce a generalized infection in an animal whose placental structure is similar to that of man; (2) its ability to produce a disease with a blood phase, thereby favoring the opportunity for transplacental passage of virus; and (3) the reliability of methods for isolation and identification of this virus. Of 33 female rabbits studied, 14 were injected with vaccinia virus 6 to 16 days prior to mating and the remainder after mating. The latter animals were grouped so that 12 were inoculated during the first trimester of pregnancy, 4 during the second trimester, and 3 during the third trimester. The dosage of virus was established at 1 ml. and ranged in titer from 10^{-4} to 10^{-6}. Blood samples, taken immediately before and from 2 to 7 days after intravenous inoculation, were cultured on the chorioallantoic membranes of embryonated eggs in order to test for the presence of the vaccinia virus. Examination of the inoculated mothers showed a rather consistent appearance of vaccinial lesions in the viscera and on the lips (Figure 8). In contrast to these findings, the cesarean delivered young, as well

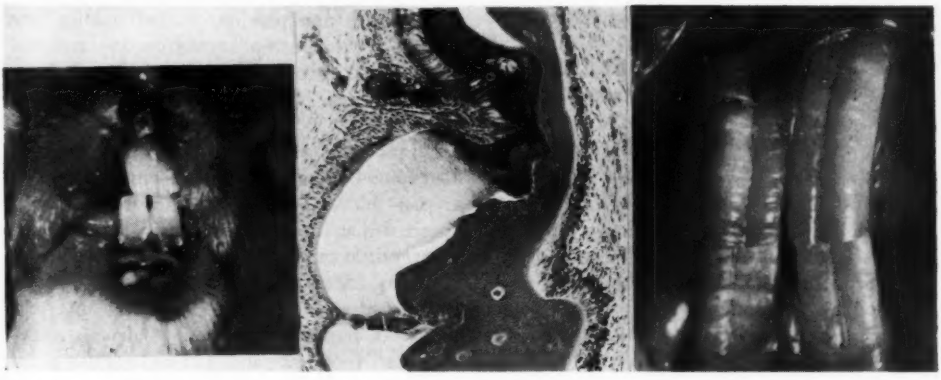

Fig. 8 Fig. 9 Fig. 10

Fig. 8.—Vaccinial lesions on the lips of a rabbit 5 days after intravenous inoculation of vaccinia virus.

Fig. 9.—Characteristic abnormalities of amelogenesis and dentinogenesis in a rabbit inoculated with vaccinia virus. Time of sacrifice: 180 days post inoculation.

Fig. 10.—Gross appearance of hypoplastic enamel defects. These become manifested at approximately 160 days following inoculation with vaccinia virus.

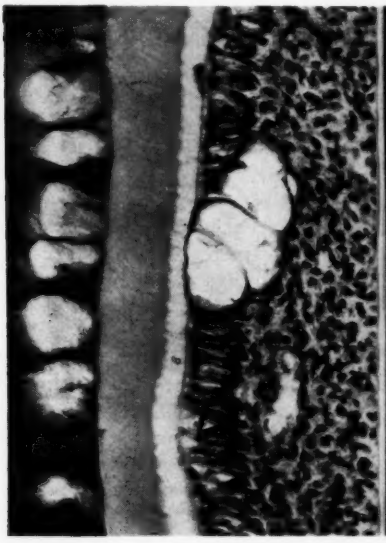

Fig. 11

Fig. 12

Fig. 11.—Enamel matrix defects and cystic degeneration of odontoblasts in the young of a mouse infected with lymphocytic choriomeningitis during pregnancy.

Fig. 12.—Typical example of exencephaly noted in the litters of rats and mice following trypan blue injection during the first trimester of pregnancy.

as those spontaneously delivered, showed no evidence of infection. A corresponding difference was also noted in the teeth; i.e., unaffected dental tissues in the offspring and a high incidence of abnormal odontogenesis in the mothers. The latter changes were of marked severity (Figure 9) and could, therefore, be recognized quite readily by gross examination (Figure 10).

In light of the correlations between maternal vaccinial infection and maternal dental defects on the one hand, and absent fetal infection and normal fetal odontogenesis on the other hand, it became of interest to study such other virus diseases that might be capable of transplacental infection.[19] Selecting lymphocytic choriomeningitis as an example, it was found in 153 inoculated mice that not only did a viremia develop in the offspring, but the fetal teeth, as well as the adult teeth, showed occasional evidence of disturbed odontogenesis[20] (Figure 11).

4. Trypan blue.

Although the previously cited studies have shown a relationship between many maternal disease processes and congenital dental abnormalities, little is known of the mechanism by which these defects are produced. Demonstrating such abnormalities as hydrocephalus and spina bifida in litters of rats inoculated with trypan blue, previous investigators[21] hypothesized that tissue breakdown in maternal diseases causes a release into the circulation of abnormal metabolite particles which bind with various plasma proteins. Evaluating this concept with particular reference to dental tissues, a series of 50 rats and 102 mice were given one or two subcutaneous injections of 0.25 to 1.0 cc. of one percent trypan blue. The time of inoculation was varied from the pre-breeding period to the ninth day of pregnancy. Additional rats and mice were similarly injected with distilled water in order to serve as controls. Although an appreciable number of abnormalities,

principally exencephaly, appeared in the young of the experimental groups (Figure 12) no disturbances of tooth formation were noted. Similarly, the continually developing incisors of the injected mothers also showed no changes.

DISCUSSION

On the basis of findings described in the human material, it would seem that abnormalities of odontogenesis are far more common in deciduous teeth than heretofore suspected. It would further appear that the intrauterine period does not effectively insure against dental injury.

With reference to the experimental observations on rats, no definite statement can be made regarding the pathogenesis of dental defects in the pyrexic group. Seemingly, however, they represent either an injurious response to certain altered metabolic functions induced by the fever, or a response to fever *per se*. With respect to the latter hypothesis, it should be indicated that no temperature recordings were made of the fetuses in utero.

In the alloxan diabetic group of animals, it is interesting to speculate whether the dental changes in the parent animals could be attributed to dysfunction of the pancreas with its associated hyperglycemia, or to drug toxicity alone. Similarly difficult to account for are the dental abnormalities in the young of alloxan injected mothers. However, in light of the apparently normal pancreatic tissue in these animals, it is probable that the developmental dental defects are related to the maternal diabetes.

The absence of dental defects in the young of rats infected during pregnancy with vaccinia is not surprising in light of the apparent inability of the virus to produce a transplacental fetal infection. On the other hand, it might be anticipated that a virus capable of transmitting infection from maternal organism to fetus could cause congenital tooth defects. Whether the dental findings presented in the lymphocytic choriomeningitis group would have been more consistently abnormal if a predominantly viscerotropic virus had been used is, of course, conjectural.

With reference to the negative maternal and fetal dental findings in the groups of rats and mice injected with trypan blue, it is unlikely that the time of pregnancy, when dye was given, could have been a factor in sparing the offspring. Rather, it could be assumed that the dye induced a type of metabolic disturbance that was related to nondental types of malformations.

CONCLUSIONS

On the basis of the presented review of clinical and experimental studies, it is apparent that the developing dental tissues of young in utero may be significantly affected by maternal disease. However, it should be emphasized that a defect of development can only involve such organs or tissue sites that are incompletely formed at the time of maternal disturbance. It is also recognized that certain injurious agents have a selective effect on only specific tissues. Either of these situations may account for the variable incidence of congenital dental defects described in the present report.

NATIONAL INSTITUTE OF DENTAL RESEARCH, U.S. Dept. of Health, Education, and Welfare, Bethesda, Maryland.

BIBLIOGRAPHY

1. Stein, G.: Enamel damage of systemic origin in premature birth and diseases of early infancy. Am. J. Orthodontics and Oral Surgery 33: 831, Dec. 1947.
2. Sheldon, M., Bibby, B., and Bales, M.: The relationship between microscopic enamel defects and infantile debilities. J. Dent. Res. 24: 109, April 1945.
3. Wolfe, J.: Teeth in fetal rickets. Am. J. Diseases Child. 49: 905, April 1935.
4. Diamond, M. and Weinmann, J.: The Enamel of Human Teeth. Columbia University Press. 1940.
5. Mellanby, H.: The effect of maternal dietary deficiency of vitamin A on dental tissues in rats. J. Dent. Res. 20: 489, Oct. 1941.
6. Kreshover, S., Clough, O., and Bear, D.: A study of prenatal influences on tooth development in humans. J. Am. Dent. Assn. 56:230, Feb. 1958.
7. Kreshover, S.: Histopathologic studies of abnormal enamel formation in human teeth. Am. J. Orthodontics and Oral Surgery 26: 1083, Nov. 1940.
8. Sarnat, B. and Schour, I.: Enamel hypoplasia (chronologic enamel aplasia) in relation to systemic disease. J. Am. Dent. Assn. 28: 1989, Dec. 1941.
9. Mellanby, M.: Diet and the teeth: an experimental study. Medical Research Council, Spec. Rep. Ser., London, No. 140, 1929; No. 153, 1930.
10. Berman, M., Edwards, L., and Kitchin, P.: Effect of artificially induced hyperpyrexia on tooth structure of the rabbit. Proc. Soc. Exp. Biol. & Med. 41: 113, May 1939.
11. Bevelander, G. and Bernstein, J.: The effect of artificially induced fever on the structure of the developing teeth of the rat. J. Dent. Res. 19: 155, April 1940.
12. Kreshover, S. and Clough, O.: Prenatal influences on tooth development. II. Artificially induced fever in rats. J. Dent. Res. 32: 565, Aug. 1953.
13. White, P.: Pregnancy complicating diabetes. Surg., Gyn., Obst. 61: 324, Sept. 1935.
14. Ziskin, D. Siegel, E., and Loughlin, W.: Diabetes in relation to certain oral and systemic problems. J. Dent. Res. 23: 317, Oct. 1944.
15. Brauer, J. and Bahadar, M.: Variations in calcification and eruption of the deciduous and the permanent teeth. J. Am. Dent. Assn. 29: 1373, Aug. 1942.
16. Kreshover, S., Clough, O., and Bear, D.: Prenatal influences on tooth development. I. Alloxan diabetes in rats. J. Dent. Res. 32: 246, April 1953.
17. Evans, M. W.: Congenital dental defects in infants subsequent to maternal rubella during pregnancy. Med. J. Australia 2: 225, Sept. 1944.
18. Kreshover, S., Clough, O., and Hancock, J.: Vaccinia infection in pregnant rabbits and its effect on maternal and fetal dental tissue. J. Am. Dent. Assn. 49: 549, Nov. 1954.
19. Lillie, R. and Armstrong, C.: Pathology of Lymphocytic choriomeningitis in mice. Arch. Path. 40: 141, Sept. 1945.
20. Kreshover, S. and Hancock, J.: The effect of lymphocytic choriomeningitis on pregnancy and dental tissues in mice. J. Dent. Res. 35: 467, June 1956.
21. Gillman, J., Gilbert, G., and Gillman, T.: Preliminary report on hydrocephalus, spina bifida and other congenital anomalies in the rat produced by trypan blue; the significance of these results in interpretation of congenital malformations following maternal rubella. South African J. Med. Sc. 13: 47, March 1948.

CONGENITE DEFECTOS DENTAL

Dr. chir. dent., med., e phil. Seymour J. Kreshover

SUMMARIO IN INTERLINGUA

Defectos disveloppamental del dentes ha longemente essite le thema de studios clinic e experimental. Nonobstante, in quasi omne casos le relationes etiologic inter varie disordines systemic e iste anormalitates ha essite establite con referentia al periodo postnatal. Il es dunque non surprendente notar le quantitate limitate de informationes disponibile in re le thema de influentias prenatal super le formation del dentes. Le pauc reportos trovabile in le litteratura include illos de Stein, qui reportava 8 casos de hypoplasia del email in unertio—i.e., le incisores —ex le dentes decidue; de Sheldon, Bibby, e Bales, qui notava defectos hypoplastic de origine intrauterin in 5 individuos ex un gruppo de 34 individuos studiate; de Wolfe, qui notava le signification de rachitismo materne pro le disveloppamento dental; e de Diamond & Weinmann, qui studiava le signification de syphilis materne pro le dentes. In studios experimental Mellanby notava incisores e molares de formation anormal in le prole de rattos que habeva essite mantenite super dietas deficiente in vitamina A ante lor copulation.

Super le base del revista hic presentate de studios clinic e experimental, il es evidente que le disveloppante histos dental del prole in utero pote esser afficite significativemente per morbos materne. Nonobstante, on debe sublinear que un defecto de disveloppamento pote afficer solmente le organos o sitos de histos que non as completemente formate al tempore del disordine materne. Il es etiam recognoscite que certe agentes nocive ha un action selective super solmente histos specific. Iste duo considerationes pote explicar le variabilitate del incidentia del congenite defectos dental que es describite in le presente reporto.

SURGICAL PROCEDURES IN PERIODONTAL THERAPY

HARRY ROTH, D.D.S.*

HOWARD L. WARD, D.D.S.*

Periodontal therapy resolves itself, according to Miller and Sorrin into three main objectives:

1. The removal of the etiological factors
2. The re-establishment of tissue health and function
3. The institution of procedures for the maintenance of health and tissue resistance.

As the title suggests, this article will be limited to the surgical phase of periodontal therapy. Unanimity as to the perfect technique in periodontal therapy has not been achieved as yet. The choice of therapy depends upon what procedures are indicated in that particular case. Recently dentistry has witnessed a rebirth of surgical procedures with many variations. This has led to an existing confusion in the literature.

The objective of periodontal therapy is to create a more favorable periodontal environment so that the patient can accomplish and maintain proper oral physiology so vital in maintaining the health of the periodontal tissues. In order to effect results such as elimination of pockets, restoration of gingival form, and regeneration of new bone, it may be necessary to institute a combination of various phases of therapy. These phases are subgingival curettage and root planing, occlusal adjustment, diet evaluation and guidance, habit evaluation and substitution, teaching of home care, and evaluation and performance of the necessary surgery. Surgery therefore, in itself is not a cure but an adjunct in achieving the proper case result and should be performed following coronal and root scaling.

Some of the accepted terms used in periodontal surgery follows: Let us first define curettage:

Curettage consists of the debridement of the inner wall of the gingivae, removing the proliferative, ulcerative epithelium, as well as the inflammatory tissue subepithelially. Scaling however, may be reserved for the removal of calculus and the planing of the root surface. The indications for gingival curettage are:

1. the elimination of gingival hyperemia and cyanosis.
2. pockets suitable for attempts at reattachment.

Flap operation, formerly known as a Robiscek operation, introduced in the United States by Zentler. This operation has many variations. However, they all have one common purpose: to expose the underlying diseased tissue in order to facilitate its thorough removal. The flap offers greater access in order to accomplish and facilitate curettage. Some of the former and present modified flap procedures are:

A. Kirkland-flap, which exposes the tortuous pocket and granulomatous tissues by separating the gingival papillae and flapping the tissue back without vertical incisions. Interproximal sutures are usually indicated.

B. Robiscek and full flap operation, consists of vertical incisions, mesial and distal to the involved area, usually one quadrant. The flap of soft tissue is retracted. The alveolar bone is exposed. Curettage and debridement of hard and soft tissues are completed. The gingival margin is contoured and sutured back to position. The full flap differs from the Robiscek in that bony deformities may be corrected.

C. Modified flap operation—is a modification of the full flap operation exposing the root and alveolus thus offering limited accessibility. In this operation there are no vertical incisions. When excessive tension is placed on the retracted gingivae and mucosa, the full flap operation is indicated.

D. Miller Button-Hole Incision—consists of a vertical incision apical to the marginal gingiva

* Assistant Professor of Periodontia and Oral Medicine, New York University, College of Dentistry.

overlying the area of the infected pocket. The marginal gingiva is left intact to minimize any possible post-operative recession. Debridement of soft tissue and root surface is done through the incision.

E. Sorrin Semi-Lunar Incision—is made coronal to the anticipated base of the periodontal pocket. The marginal gingiva is left intact and debridement of granualomatous tissue and root planing is accomplished by way of a semi-lunar incision. Two sutures help to retain the tissues in place.

Gingivectomy—the surgical removal of unsupported gingiva to the point where it is firmly attached, thus establishing a new gingival margin more apical to the old with a shallow or non-existing sulcus. Indications for gingivectomy are pockets where the soft tissue portion is densely fibrotic and will not regress to a desired architectural form or heal without a deepened gingival sulcus. In addition, where the gingival tissue is so thin that when the curettage is done, the curette perforates the gingival wall. Surgery should also be performed in deep gingival or periodontal pockets which persist after therapy.

Gingivoplasty — the surgical removal of gingival deformities following subgingival curettage in order to create more favorable physiologic contours. Gingivoplasty usually accompanies gingivectomy. Indications are the removal of enlarged gingival margins following gross interproximal tissue loss and varying heights of gingival margins.

Osteoplasty—the recontouring of bone which does not involve the removal of supporting bone.

Osteoectomy—the removal of alveolar bone from about the root of a tooth in order to eliminate an adjacent pocket. The amount of bone removed should be consistent with the retention of the tooth in question. Osteoectomy is indicated for correction of interproximal bony craters; shallow intrabony pockets; bifurcation and trifurcation involvments.

Intrabony pockets—where the base of a pocket is apical to the crest of the bone. The decision as to which surgical procedure is to be performed in the treatment of the intrabony pocket depends on a careful evaluation of the situation. The depth and width of intrabony pockets are important determinants for therapy. Extremely deep, narrow pockets constitute a crypt-like defect in the bone. This offers the advantage of the protection of the blood clot after therapy and the prevention of the shrinkage of the gingiva to any appreciable degree. Depth, width, crestal form and even adjacent tissue will often determine whether a re-attachment attempt is justified or whether osteoplastic reshaping will be a more effective way in which to eliminate the intrabony character of the lesion. All known etiologic factors must be eliminated prior to the soft and hard tissue debridement. The treatment of the intrabony pocket may be done by the curettage-gingivectomy method, or the flap approach.

Vestibule Resections — or extensions — deepening of the fornix of the vestibule is usually associated with abnormally attached frena and periodontal pockets extending apical to the mucogingival junction and/or the fornix of the vestibule. It is handled similarly to a lower midline frenum operation.

Gingival Extension—the surgical procedure for extending or increasing the zone of attached gingiva. This operation is indicated in increasing the vestibule where intrabony, gingivectomy, gingivoplasty, osteoplasty and osteoectomy techniques are employed.

Frenotomy—the surgical separation of the abnormally attached frenum in order to free the pull on the gingival margin and increase the depth of the vestibule.

The judgement of the practitioner, based on his experience and the requirements of the case, will determine which technique will be most successful. Many techniques are used in one operation and are only enumerated for purposes of definition and classification. Prichard states that Goldman's prinicpal of gingivoplasty pervades all periodontal surgical techniques and it was the repeated failures with the gingivectomy techniques that led Schluger to think in terms of osseous surgery.

Goldman, Schluger, and Fox agree with

Glickman and others that gingivectomy must be considered the basis of the surgical elimination of pockets. The "plastic" type operation is chiefly concerned with anatomic form as it relates to physiologic function for long range preservation and maintenance of the health of the periodontium.

As the gingivectomy is the basis of surgical technique, subgingival curettage and root planing must be considered the basis of periodontal therapy. In most cases root planing and subgingival curettage should be completed and the case re-evaluated before surgical intervention.

There are many advantages in having subgingival curettage and root planing precede gingival and osseous surgery.

1. It permits the patient to be psychologically prepared for surgery.

2. Surgery in any form is a traumatic experience for those even in good health.

3. Performance of subgingival curettage initially gives the practitioner sufficient time to observe possible deviations in healing.

4. The tissue response following surgery is augmented, when previous subgingival curettage, root planing, equilibrium and oral physiotherapy is completed.

5. The time consumed in presurgical procedures permits the practitioner to give consideration to the general physical status of the patient, being mindful of complicating systemic factors which in turn may interfere with

SURGICAL PROCEDURES IN PERIODONTAL THERAPY

Type	*Indications*	*Contra Indications*
1. Gingivectomy Surgical removal or excision	1. Dense fibrotic gingival tissue 2. Deep pockets which persist after subgingival curettage and root scaling 3. Bifurcation and trifurcation involvements	1. 1. Poor Physical health 2. Esthetic consideration especially of the anterior teeth 3. Presence of acute infections as acute necrotizing ulcerative gingivitis or herpetic-ulceromembranous stomat.tis 4. Emotionally disturbed patient
2. Gingivoplasty	1. Enlarged gingival margins: following gross interproximal tissue loss 2. Varying heights of gingival margins	2. 1 & 3 same
3. Osteoectomy	1. To help eliminate shallow intrabony pockets 2 To correct interproximal bony craters 3. To correct bifurcation and trifuration involvements	3. 1 & 3 same
4. Osteoplasty	1. To help create better bone contour	4. 1 & 3 same
5. Flap approach a. Miller Button-hole b. Sorrin Semi Lunar c. Kirkland d. Robiscek e. Modified flap f. Full flap	Type of operation to be determined by existing conditions 1. following acute abscess 2. presence of sinus openings of fistulous tracts 3. In tortuous, narrow pockets 4. In anterior region, where esthetics may be an important factor 5. In intrabony pocket 6. For ease, accessibility, and visability 7. As an exploratory operation—to determine prognosis of situation	5. 1 & 3 same
6. Vestibular Extensions Gingival Extensions Frenotomy	1. To extend or increase zone of attached gingivae a. in intrabony pockets b. gingivectomy or gingivoplasty techniques 2. To rebase the pull of membranous or prena attachments which exert tensions on free gingival margins or create interferences.	6. 1 & 3 same

healing and resistance to infection.

The above factors associated with surgery may precipitate a nutritional deficiency in patients whose nutrition is already in a state of tenuous equilibrium. Some of the factors which may influence the patient's state of nutrition are:

1. Emotional stress—in anticipation of, and during surgery;

2. Blood volume loss from 50-400 mg. as reported by Johnson;

3. The inability to properly chew post-operatively for two to four weeks may also influence malnutrition. Therefore supplementation and special dietary advice should be given careful consideration.

Paul R. Stillman, in teaching equilibration of occlusion stressed good anatomic form. Time and again he stressed health and not disease. Stillman felt that by creating the proper environment by proper form, whether of the hard or soft tissue structure of the oral cavity, nature would rise to the occasion and health would prevail. This same philosophy may be applied to all periodontal therapy.

This paper, limited in scope, may have neglected to mention many men of national and international prominence who have contributed so fruitfully to the advancement of the treatment of periodontal disease. It both recognizes and appreciates their contributions. The paper has merely attempted to clarify the main differences in the numerous existing techniques.

BIBLIOGRAPHY

Ariaudo, A. and Tyrrell, H. A.: Repositioning and increasing the zone of attached gingivae. J. of Perio., Vol. 28, No. 2, April, 1957.

Beube, F. E.: Periodontology. The Macmillan Co. New York, 1953.

Friedman, N.: Periodontal osseous surgery: osteoplasty and osteocetomy. J. of Perio. Vol. 26, pp. 257-69, 1955.

Glickman, I.: The results obtained with an unembellished gingivectomy technic in a clinical study in humans. J. of Perio., Vol. 27, No. 4., Oct., 1956.

Glickman, I.: Clinical Periodontology, W. B. Saunders Co. Phila., 1953.

Goldman, H. M.: The development of physiologic contours by gingivopasty. Oral Surg., Oral Med., and Oral Path., Vol. 3, p. 879, 1950.

Goldman, H. M.: Schluger, S. and Fox, L. Periodontal Therapy, p. 227, C. V. Mosby Co., St. Louis, 1956.

Johnson, R. L.: Blood loss in oral surgery. J.D.R. 35: #2, p. 179, April, 1956.

Kirkland, O. in Miller, S.C.: Textbook of Periodontia, Third Edition. The Blakiston Co. Phila.

Miller, S. C.: Textbook of Periodontia, Third Edition. The Blakiston Co. Phila.

Ochsenbein, D.: Osseous resection in periodontal surgery. J. of Perio., Vol. 29, No. 1, Jan., 1958.

Prichard, J. F.: Various techniques in periodontal case management. The Texas Dent. J. Feb., 1955.

Ravidin, I. S. and Zintel, H. A.: Nutrition in the care of the surgical patient, p. 84 in Wohl, M. G., and Goodhart, R. S., modern nutrition in health and disease. Lea and Mebiger, Phila. 1955.

Schluger, S.: Osseous resection—a basic principal in periodontal surgery. Oral Sur., Oral Med., and Oral Path. Vol. 2:316, 1949.

Sorrin, S.: The use of the toothbrush as a preventive and cure. Dental Cosmas, April, 1931.

Stillman, P. R.: Notes from lectures.

PROCEDIMENTOS CHIRURGIC IN LE THERAPIA PERIODONTAL

Dr. chir. dent. Harry Roth e Dr. chir. dent. Howard L. Ward

SUMMARIO IN INTERLINGUA

Le therapia periodontal, secundo Miller e Sorrin, ha ultimemente tres objectivos principal:

1. Le elimination del factores etiologic.
2. Le restauration del sanitate e function del histos.
3. Le institution de methodos pro le mantenentia del sanitate e del resistentia del histos.

Multe avantages resulta del effectuation de curettage subgingival e de planage de radice ante le execution de chirurgia gingival e ossee:

1. Illo prepara le patiente psychologicamente al operation chirurgic.
2. Interventiones chirurgic de omne genere es un experientia traumatic mesmo pro subjectos in bon sanitate.
3. Le effectuation initial de curettage subgingival provide le intervallo desirabile pro observar deviationes possibile in le processo resanatori.
4. Le tempore usate in manovras prechirurgic permitte al operante prestar attention al general stato physic del patiente e al possibile factores systemic de character complicatori le quales, de lor parte, pote obstruer le processo resanatori e le resistentia a infectiones.

Le objectivo del therapia periodontal es crear un plus favorabile milieu periodontal, de maniera que le patiente pote establir e mantener le appropriate physiologia oral que es si importante pro le preservation de san histos periodontal. Pro effectuar resultatos como elimination de bursas, restauration del forma gingival, e regeneration de osso, il pote devenir necessari instituer un combination de varie phases de therapia. Iste phases es curettage subgingival e planage de radice, adjustation occlusional, evalutation del dieta e recommendationes correspondente, evalutation del habitudes del patiente e suggestiones pro lor modification, instruction in mesuras que le patiente debe prender in su vita private, e evalutation e effectuation del requirite operation chirurgic. Le operation chirurgic per se non es un cura sed un adjuncto in le effectuation del desirate resultato. Illo deberea esser executate post desquamation del corona e del radice.

Case Report

AMELOBLASTOMA OF THE MANDIBLE

HARRY FREDRICS, B.A., D.D.S.*
DAVID L. HEMPHILL, B.A., D.D.S.†
IRWIN W. SCOPP, B.S., D.D.S.[3]‡

CHIEF COMPLAINT

R. P., a 29 year old Puerto Rican male, was referred to the Veterans Administration Hospital, New York, New York from the New York Regional Office with a diagnosis of a "cyst in the mandible". The patient was admitted on December 17, 1957 for an enucleation and biopsy of this lesion.

The patient was well-developed, well-nourished, and was in no acute distress at time of admission. The history revealed that the patient had had an extraction of the mandibular right first molar approximately 18 years prior to admission or at age 11. One month prior to admission, routine dental X-rays revealed an area of pathology in the right side of the mandible in the first molar area. The patient reported no pain in the involved area, the teeth were not sensitive to percussion nor was there any history of inflammation.

HISTORY

The personal history was essentially negative with the patient claiming no significant childhood nor adult illnesses other than tonsillitis in 1952. The patient smoked one pack of cigarettes per day and drank alcohol occasionally but he denied any present drug medication. The patient also gave no history of previous operations or serious injuries.

PHYSICAL EXAMINATION

The physical findings were unremarkable with no pathology observed.

ORAL EXAMINATION

The regional examination of the oral cavity disclosed dry lips with normal color and pigmentation. The general oral hygiene was fair to good. The mucous membrane of the buccal mucosa, vestibule, palate and floor of the mouth were essentially negative. The tongue was well papillated, non-indurated and not coated. The patient had a well fitting maxillary partial denture. The mandibular teeth were in a fair state of repair with the second mandibular right molar inclining mesially toward the edentulous area of the extracted first right molar. In this area there was a slight alveolar swelling on the buccal side. The lingual surface was essentially negative. The area was not sensitive to percussion. No crepitus was noted.

LABORATORY TESTS

Laboratory tests of urine and blood including calcium and alkaline phosphatase levels, were all essentially normal.

SURGICAL PROCEDURE

On December 18, 1957 the patient was taken to the operating room (Figs. 1, 4, 5) after being carried on Terramycin 250 mg. every six hours for two days. For premedication, the patient was given Nembutal 100 mg., Atropine Sulfate 0.0004 gm. and Demerol 100 mg. one

* Staff Oral Surgery, Dental Service, Veterans Administration Hospital, New York City, Instructor in Oral Surgery, Department of Oral Surgery, New York University, College of Dentistry, N. Y. C.

† Resident in Oral Surgery, Veterans Administration Hospital, New York, New York.

‡ Chief, Dental Service, Veterans Administration Hospital, First Avenue at East 24th Street, New York 10, New York. Associate Clinical Professor of Periodontia and Oral Medicine, New York University College of Dentistry, New York, New York.

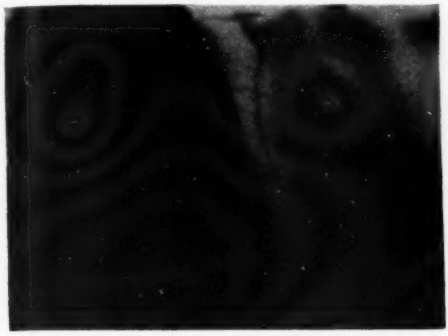

Fig. 1. The periapical roentgenogram of the lower right first molar area. The triangular outline and the small bony process adjacent to the second molar suggest that the lesion may be polycystic or multiple in nature.

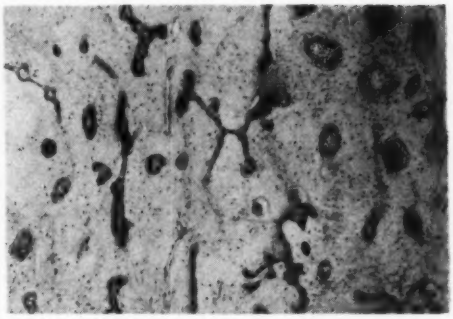

Fig. 2. A low power microphotograph of the ameloblastoma. The loose connective tissue is seen with invasion by small cords and tube-like masses of epithelioid tissue.

hour prior to surgery. In the operating room, the patient received a nasotracheal intubation and was maintained on nitrous oxide and oxygen following intra-venous sodium pentathol 0.5%. The face and neck were prepared with phisohex followed by aqueous zephiran chloride and the patient was draped for an oral surgical procedure. After the throat pack consisting of a saline moistened two inch gauze was placed, an incision along the gingival margin of the buccal mucosa was made. A flap extending from the mandibular right first premolar distally to the second molar provided adequate access to the area of pathology. The entire mucoperiosteum was detached from the bone. With a rongeur, a thin plate of bone was removed from the greatest area of buccal expansion. As the cyst-like area came into view, it was dissected away from the bone by the use of a curette. The attachment from the bone was severed without difficulty and the entire lesion was removed and sent to the pathology laboratory, for microscopic examination. The grayish-white mass was removed in toto and measured approximately 1.5 x 1.0 x 1.0 cm. With a bone file, the rough edges of bone were smoothed. Next the cavity was debrided using warm saline and then the mucoperiosteum was sutured in place with 000 silk. The throat pack was removed after aspirating the oral cavity and the patient was extubated on the table. There were no post-operative complications as the mucous membrane of the area of surgery was essentially negative one week later. The patient was discharged on December 23, 1957. The sutures were removed on December 26, 1957 and the patient was advised to return monthly for follow-up examinations.

Biopsy Report—(Figs. 2, 3.)

"Gross Description: Submitted in Bouin's solution, specimen consisted of an oval shaped mass of tissue lined by a smooth translucent capsule. The cut surfaces are firm gray-white and homogeneous."

"Microscopic Examination: Sections show a light loose connective tissue infiltrated with numerous small masses, cords and tube-like structures. The cells forming the noted structures are very smooth and hyperchromatic but reveal no malignant changes. The section is lined almost completely by a few layers of a keratinized epithelium."

"Diagnosis: Adamantinoma."

PATHOGENESIS

The adamantinoma, or ameloblastoma as it is more generally called, is derived from ectodermal epithelium or the epithelial lining of a dentigerous or radicular cyst. According to Thoma,[1] "the tumor may form from cells that have already become differentiated or from embryonic epithelial cells." To quote further, Thoma states, "The tumor may be derived from

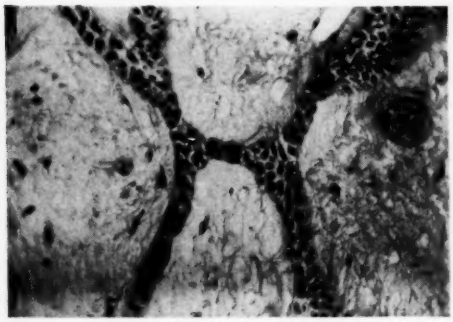

Fig. 3. The appearance of the epithelioid cords under high power magnification. There is a striking resemblance to the early stages of tooth bud formation.

Fig. 4. The appearance of the operative site two months later. The bony conformity with the original polycystic lesion is evident.

(1) the genetic disturbance of the enamel organ or the tissue forming it, (2) cell rests of the enamel organ, either during tooth development or later, (3) the epithelium of odontogenic cysts, (4) the surface epithelium covering the jaw, and (5) displaced epithelium in the other parts of the body."

CLINICAL FINDINGS

It is more common for an ameloblastoma to be found in the mandible than the maxilla and it is more frequently found in young adult males than in females, the average age being 25 to 30 years. If found in the mandible, the lesion is more likely to be associated with the molars or premolars. The lesion expands from within resulting in a buccal or lingual plate of parchment-paper thinness. Generally, the cystic type develops by central necrosis and liquefaction of the solid type.

ROENTGEN FINDINGS

Thomas reports that "there is no consistant characteristic roentgen signs in ameloblastoma. The monocystic types resemble odontogenic cysts, the polycystic types may be mistaken for other tumors that form multiple lesions. For differential diagnosis, multilocular cysts, giant cell tumor, fibroma, fibrosarcoma, myxoma and metastatic carcinoma must be considered."

MICROSCOPIC FEATURES

Microscopic features, as quoted from Anderson, imply a great variety of cell patterns. "Columns and aggregations of epithelial cells occur in various stages of differentiation. There may be solid nests of cords of cuboidal cells, or they may be arranged in a glandular pattern. Cylindrical cells resembling ameloblasts, occasionally accompanied by flat cells, surround a stellate reticulum, resembling the enamel organ. . . . The stellate reticulum may degenerate, giving rise to cysts. No enamel formation has ever been observed in these tumors."

TREATMENT

There seems to be varied schools of thought on the treatment of an ameloblastoma. Conservative treatment consists of complete enucleation of the cystic area. Some advocate radical

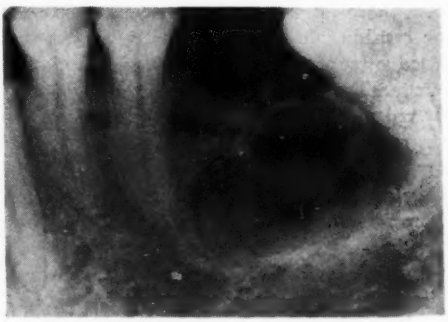

Fig. 5. The post-operative appearance four months after operation. The cavity is almost completely obliterated by infiltration with bone.

surgery into healthy bone to help prevent recurrence or development into adenoacanthoma, squamous cell carcinoma or, rarely, adenocarcinoma. Others have had success with radiation or formalin application to the bone lesion following conservative surgery. As is true with all surgery on tumors or neoplastic growths, there is no all inclusive method of treatment. Great consideration must be given to the specific case.

BIBLIOGRAPHY

1. Thoma, K. H.: Oral Pathology, ed. 4, St. Louis, Mo. The C. V. Mosby Co., 1954.
2. Anderson, W. A. D.: Pathology, ed. 2, St. Louis, Mo. The C. V. Mosby Co., 1953.
3. Boyle, P.: Kronfeld's Histopathology of the Teeth and Their Surrounding Structures. Lea and Febiger, Philadelphia, 1952.

JOURNAL OF DENTAL MEDICINE INCREASES SUBSCRIPTION RATES

The subscription rate of Dental Medicine will increase commencing with the January 1959 issue. The regular domestic rate will be $6.00 per year; Canadian subscribers $6.25; and Foreign subscriptions will be $6.50 per year. Single copies of the Journal will cost $1.50 each. A special student subscription rate will be $4.00 annually. Members of the Academy of Dental Medicine will not be affected by this rate increase since an annual subscription is included in the membership dues.

Book Reviews

Oral Roentgenographic Diagnosis by Edward C. Stafne, D.D.S., F.A.C.D. Philadelphia, W. B. Saunders Co., 303 pages, 1138 Illustrations on 423 figures, 1958, $14.50.

In the preparation of this text Dr. Stafne has been fortunate to have at his disposal roentgenographs that have been accumulated over a period of 33 years, the majority of them concern patients suffering from systemic disease. The author has had the opportunity to observe and study oral roentgenograms of many varied disease entities and has demonstrated that the roentgenographic patterns of the jaws are as frequently diagnostic of systemic disease as are lesions of the oral soft tissues.

The subject matter covered includes anatomic landmarks, anomalies and condition peculiar to the teeth themselves, pathologic calcifications of the hard and soft tissues, infections, cysts and tumors of the jaws, and oral roentgenographic manifestations of systemic disease. In addition to identifying a condition that is revealed by the roentgenographic examination, the author has discussed its significance and in some cases, has offered suggestions as to its treatment.

The text is well written and excellently illustrated. The type is large and clear and is printed on a high grade of glossy paper. There is an adequate bibliography at the conclusion of each chapter.

Dr. Stafne's book is a welcome addition to the dental literature.

Human Embryology, by Bradley M. Patten, Second Edition, Blakiston Division, McGraw-Hill Book Company, New York.

This new edition is a tribute to the author's clarity, conciseness and completeness of his work in human embryology. The chapters include: Reproductive Organs; Gametogenesis; Sexual Cycle and Fertilization; Cleavage, Germ layers, and Establishment of Embryonic Body; Early Differentiation of the Body and Establishing of Organ Systems; Fetal Membranes and Placenta; Age, Growth and Changes in External Form of Body; Twins, Double Monsters and Teratology; Integumentary System; Connective Tissues and Skeletal System; The Muscular System; Development of the Nervous System; The Sense Organs; Development of the Face and Jaws and the Teeth; Development of the Digestive and Respiratory Systems; Body Cavities and Mesenteries; etc.

The format of the book remains the same but recent advances have been brought to the fore, especially the work of Hertig and Rock in securing younger stages of human embryos than any heretofore known. Likewise, the early stages of human implantation made possible by these workers enabled the author to elaborate those embryologic stages in this work. Then, too, extensive changes were made in the discussion of the heart and great vessels, especially with the amazing progress made in the surgical management of congenital cardiovascular defects.

The book is a credit to both author and publisher and makes a handy reference work not only for the student but for the practitioner as well.

I. NEWTON KUGELMASS, M.D.

Tooth Replacement Through Crown and Bridge and Partial Prosthesis, by Christian Koller, Wilhelm Maudrick Publishers, Vienna, 1954, (in German) $19.20.

This third edition of a text on tooth replacement, dealing with every conceivable aspect of the discipline, enjoys wide acceptance in Europe. The brief review of biology of the supporting structures and methods for preparation by grinding of the occlusally deformed and periodontally involved teeth for restorations is inadequate. This inadequacy in dealing with the effects of developmental defects and those following non-replacement of extracted teeth, however, is well compensated for. The many-faceted review of crown, bridge and partial denture methods recommended through the years and the ingenuity used in devising many methods for the various situations encountered in over 200 cases is presented.

Of interest to this reader is the recommended means for the replacement of even one missing tooth by the use of one fixed and one movable joint abutment. As in other replacements, some of the mechanical devices are unnecessarily complicated. Three-quarter crowns are generally the abutments of choice and crowns held by posts are included in most fixed bridges with frequent combinations of varied abutments in the same replacement. Many ingenious, but complicated, clasp designs are suggested to break stress and protect "weak" remaining teeth, yet principles of splinting are not employed to stabilize loose teeth or to prevent future loosening. In fact, even in fixed bridgework involving an entire arch, at least three separate units are the author's choice.

Impression taking, bite registration, esthetic considerations of partial dentures, together with a complete consideration of various metals and their processing are worthy of note. A review of various European and American methods of crown and bridge construction with the laboratory methods involved are clearly presented. These should be of great interest and value to any general practitioner or laboratory technician.

The author accomplishes his purpose in this encyclopedic volume of giving the theory and the practically applicable methods of crown, bridge and partial denture restorations. All the techniques described were actually constructed by him to insure both his understanding of the methods and clarify in description. The text, written in simple German and copiously illustrated, can be easily understood by anyone with a minimum of German language training.

Developmental Anatomy, A Textbook and Laboratory Manual of Embryology by Leslie Brainerd Arey, Ph.D., and Robert Laughlin Rea, Sixth Edition, W. B. Saunders Company, Philadelphia.

This recent edition presents a scholarly work on embryology; Part I, gives general development; Part II, special development; Part III, laboratory manual. The entire book has been rewritten to bring it up to date not only in individual contributions but in integration of all recent achievements affecting human development. The style of writing is clear and concise, the illustrations striking and informative, the references well correlated with the body of the text. Fortunate is the student who has this Volume as a guide and mentor of his experimental embryology as a basis for his medical and dental training.

I. NEWTON KUGELMASS, M.D.

Accepted Dental Remedies, by the Council on Dental Therapeutics, of the American Dental Association, 24th edition, 224 pages, Chicago, 1959, $3.00.

Accepted Dental Remedies 1959, which bears the insignia honoring the Centennial of the American Dental Association, represents a milestone marking the twenty-fifth year in which this publication has been made available to the members of the profession. Approximately a quarter of a million copies of *Accepted Dental Remedies* have been printed during this period.

The new edition contains expanded sections on dental therapeutics. The monographs have been reviewed by the Council and its consultants and have been rewritten in the light of current scientific information. The reorganized format, which made last year's edition so popular, has been retained with minor additional improvements. A discussion of the prescription use of fluoride preparations is included in the chapter on Fluoride Compounds.

Accepted Dental Remedies 1959 contains 224 pages, including the provisions for acceptance of products, a general index, a distributor's index, an index of current reports from the Council and the Division of Chemistry, and an index of more recent reports on products not listed in the book, including those classified in Groups B, C and D.

Die Behandlung Der Gangran Der Pulpa, (The Treatment of the Gangrene of the Pulp) by Josef Vachuda, Verlag Wilhelm Maudrich, Wien, 1956. 356 pages, 353 illustrations, $14.05.

The present book represents a comprehensive survey of endodontic therapy as practiced in Germany. Of particular interest to this reader, was the discussion of the role of the non-vital tooth as a focus for various diseases. German literature has long been concerned with the relation between focal infection and systemic reaction, focal infection including not only bacterial spread but also bacterial and tissue toxins, allergens, etc. From the literature quoted in this book, unfortunately exact references are not always cited, no definite conclusion regarding this local-systemic interaction has as yet been reached by German investigators.

Dr. Vachuda comprehensively describes the mechanical, chemical and physical methods used in endodontic therapy and includes illustrations of various European instruments which are of interest.

The printing and illustrations are clear and the volume should be of value to the American practitioner interested in endodontics.

S. SIGMUND STAHL

Academy Proceedings

PHILADELPHIA SECTION

The Philadelphia Section is busy preparing for the 1960 annual meeting of the Academy and several committee meetings have been held. The hotel has been reserved and considerable thought has been given to the type of program that would be best suited for such an event. The work, so far, has been of a preliminary nature.

The annual all day meeting that has become a part of the Philadelphia Section and the Philadelphia Society of Periodontology, will take place as usual in February. This year the topic will be "Pain". These meetings have been well attended because of their informativeness. The date is February 25th, 1959. For details contact Dr. B. B. Saturen, 1930 Chestnut Street, Philadelphia, Pa. All members are cordially invited to attend.

JACOBY T. ROTHNER

MICHIGAN SECTION

In keeping with the high caliber of scientific programs set fourth in the past by the Michigan Section on the local level and the Academy on the national level, the following eminent authorities have been the guests and teachers of the Michigan Section:

1. Dr. Paul Boyle, who at our April meeting, a luncheon held during the annual meeting of the Michigan State Dental Association, gave us an insight into his philosophy of medicine and disease, when he spoke on "Oral Pathology as a Basis for Diagnosis".
2. Dr. Robert Moyer, D.D.S., M.S., a member of the staff of the Orthodontic Section of the School of Dentistry of the University of Michigan, spoke at a dinner meeting in September on "Temporomandibular Joint Problems".
3. Dr. John Gajewsti, M.D., Ph.D., was our guest in November when we held a dinner meeting during the annual Detroit Dental Review. Dr. Gajewsti is a member of the Department of Clinical Investigation of the Parke Davis Company and discussed the physiologic action of the newer drugs.

The Michigan Section was very well represented at the past annual meeting of the Academy in Montreal. It is the consensus of those who attended the meeting that a vote of thanks is in order to the members of the Montreal Section for their wonderful hospitality and for the most enjoyable and highly instructive scientific format of the meeting. It was evident that such a meeting could only have been achieved through careful planning and hard work. Again, the Michigan Section wishes to thank the Montreal Section for a terrific time.

We of the Michigan Section were both thrilled and proud to have *our* Dr. Don Lyons installed as President of the Academy for the current year. Congratulations Don, from the Michigan Section.

HERMAN M. LIFTON, *Publicity*

REPORT ON THE FIFTEENTH ANNIVERSARY MEETING OF THE AMERICAN INSTITUTE OF DENTAL MEDICINE PALM SPRINGS, CALIFORNIA, OCTOBER 12-16, 1958

DON C. LYONS, D.D.S., M.S., PH.D.

At the inviation of our Vice President Herman Becks, who is President of the American Institute of Dental Medicine and their Board of Directors, Harold Gelhaar and I attended their Annual meeting along with Reuben Feltman who is Chairman of our next Annual Academy meeting in May 1959 at Atlantic City. We were given a truly Western hospitality welcome and every day of the meeting was an extension of this welcome and cordiality on the part of Dr. Becks, his staff, and the 280 dentists and physicians that attended the meeting. We made a great many new friends.

Top: Dr. Don C. Lyons, Dr. Harold Gelhaar, Bottom: Dr. Hermann Becks, Dr. Reuben Feltman, American Institute of Dental Medicine, Palm Springs California, October, 12-16, 1958.

The educational program was outstanding. It was an inspiration and privilege to listen to such a dedicated scientist as Hans Selye who told us about the advances in the research on the relation between stress and heart disease; Emanuel Cheraskin, one of our members who will also be an essayist at our next annual meeting, who discussed his research on diabetes and on the relation between local anesthetics and blood pressure; Herbert Evans who told about the foundation work on growth hormones; K. F. Meyer who gave us new insight regarding viral diseases; Joseph C. Muhler who told of his research work in the field of caries and fluorides; and Harry Worth who gave us a new concept of what the radiograph can tell about tumors of the jaws. It was of special interest to us that Dr. Hayakawa who was lecturing on communication, language, and its meaning highly praised our Journal and its Editor for our efforts in the International spread of knowledge and understanding by publishing abstracts of our publications in Interlingua.

Dr. Feltman told the members of the Institute our plans for the Annual meeting next May, announced the essayists, and invited all to the meeting.

We all felt that this opportunity to visit the Institute and take part in its proceedings was extremely worth while and should be the beginning of an extremely advantageous interchange of guest members of two groups with the same goals; the application to dental practice the basic principals of treating the human as a whole, the fundamentals of medicine as related to the oral tissues, with resulting intelligent total treatment.

MASSACHUSETTS SECTION

The Massachusetts Section, under the able leadership of our president, Dr. Earl Stone, is planning for an active year.

Our first luncheon meeting was held at the Hotel Kenmore, Boston, on Sunday, November 2. A large attendance indicated a healthy interest in the activities of the Section and a bright forecast for future meetings.

We were honored by the official visitation of our national President, Dr. D. Chalmers Lyons, who brought us the greetings of other Sections, information about some of their projects, and the good news of the creation of new Sections.

The speaker of the afternoon was Dr. Louis Weinstein, Associate Professor of Medicine at Boston University, Lecturer on Infectious Diseases at the Harvard Medical School, Chief of the Department of Infectious Diseases at the Massachusetts Memorial Hospital, and Professor of Medicine at Tufts Medical School. He spoke on "Sedatives, Analgesics, Hypnotics, Tranquilizers, Antibiotics, and Anti-viral Drugs." Doctor Weinstein discussed these various drugs as to their pharmacology, uses, and abuses. He warned that any of the drugs in these categories, if abused, can become dangerous. He particularly mentioned the abuses of antibiotics and was able, from his own wide experience and research, to illustrate his points most effectively.

A general discussion period followed the talk and this proved helpful and interesting.

We plan to hold two further meetings during the year, one of these to be held jointly with the American Academy of General Medicine. We believe that such joint gatherings will bring about a better understanding and a closer relationship between our two professions.

MAURICE F. GROSSMAN

NEW JERSEY SECTION

The New Jersey Section held its first meeting of the fall season on October 28, 1958 at the Hotel Douglas, Newark. Dr. George Witkin, Assistant Professor of Periodontia and Oral Medicine at New York University College of Dentistry, was the essayist for the evening. He spoke on the "Appreciation of Oral Lesions," illustrating his talk with many excellent color slides. The session was a very stimulating one with the membership entering into a discussion of the diagnosis and management of the lesions illustrated.

Following adjournment of the regular meeting, a meeting was called for the 1959 convention committee. Arrangements for the scientific program are now complete and Dr. Samuel Ament had the program printed and distributed at the December meeting. Other committee chairmen reported progress in their arrangements for making the meeting in Atlantic City the best and biggest one in the history of the academy.

ABRAM I. CHASENS, *Delegate*

THIRTEENTH ANNUAL MEETING AMERICAN ACADEMY OF DENTAL MEDICINE

The Thirteenth Annual Meeting of the American Academy of Dental Medicine will be held in the Claridge Hotel, Atlantic City, N. J. on Friday, Saturday and Sunday, May 15th, 16th and 17th, 1959.

An excellent scientific program is planned for this three day meeting. Clinicians will include:

Emanuel Cheraskin, M.D.,D.M.D.
Lester R. Cahn, D.D.S.,F.D.S.,R.C.S.
Sigmund S. Stahl, D.D.S.,M.S.
Harry Blechman, D.D.S.
Austin H. Kutcher, D.D.S.
Harold J. Jeghers, M.D.
Bernard Greenberg, D.D.S.
Morris B. Auerbach, D.D.S.
George G. Stewart, D.D.S.

A detailed program of this meeting will be published in the next issue of the Journal of Dental Medicine.

DR. REUBEN FELTMAN, DR. FRANK SCHAMBACH, *General Chairmen*
DR. ABRAM I. CHASENS, *Scientific Chairman*

MONTREAL SECTION

At our first reunion in September last we had the pleasure and honour to have as guest speaker Dr. Don C. Lyons, National President. This reunion was held in the Laurentian, Jasper in Quebec, St-Donat. His subject was "Preparatory medication for the dental patient." All our members and guests were delighted to hear him and spent a wonderful week-end in this enchanting decor.

A Convention of the Canadian National Dental was held in Montreal last September. One of our past presidents, Dr. V. A. Dubé was appointed Honorary Member of the Société Dentaire de Montreal. Other nomination as "F.I.C.D.," were Drs. P. J. Gitnick, Wilfred Johnston, Conrad Godin of Trois-Rivières, and J. McCutcheon, Dean, Faculty of Dentistry, McGill University, Montreal. Dr. Lou Rosen of Montreal was named "F.A.C.D."

GUY LANGELIER, *President*

DR. SAMUEL CHARLES MILLER MEMORIAL LECTURE

The first Dr. Samuel Charles Miller Memorial Lecture will take place on Tuesday, February 10, 1959 at 8:00 P.M. in the new building of the New York University College of Dentistry located at 421 First Ave., New York, N. Y. The entire dental profession is cordially invited to attend.

SIDNEY SORRIN, *Chairman*

Announcements

POSTGRADUATE TRAINING IN PERIODONTIA AND ORAL MEDICINE NEW YORK UNIVERSITY COLLEGE OF DENTISTRY

The following postgraduate courses in Periodontia and Oral Medicine will be offered at the New York University College of Dentistry and will be given by Dr. Sidney Sorrin and associates:

No. 124. REFRESHER COURSE IN PERIODONTIA AND ORAL MEDICINE

Ten sessions, Wednesday, 4:00 to 6:00 P.M. February 11, 1959 to April 22, 1959.
Tutition and books, $156.00. Instruments approximately $55.00.

Theoretical and practical application of periodontal diagnosis and techniques to general dentistry. Four lecture and six clinical demonstration sessions. Equilibration of occulsion is performed by the student on casts. Patient demonstrations include equilibration of occulsion, conservative and radical therapy, home care, etc.

No. 123. PERIODONTIA AND ORAL MEDICINE IN THE SPANISH LANGUAGE

Four weeks, Monday through Friday, 9:00 A.M. to 4:00 P.M.
February 23 to March 20, 1959 inclusive. Tuition including books and instruments approximately $385.00.

Theoretical and practical application of Periodontia and Oral Medicine is fully explained and demonstrated. Equilibration of occlusion is performed by the student on casts. Lectures and clinical demonstrations of diagnosis and treatment planning, conservative and radical techniques, equilibration of occlusion, home care, etc. The entire course is conducted in the Spanish language.

No. 122. COMPREHENSIVE PREPARATION IN PERIODONTIA AND ORAL MEDICINE

No. 122 A. One academic year, full time. September 17, 1958 to May 22, 1959.
Tuition $1,010.00. Instruments and books approximately $135.00.

No. 122 B. Two academic years, full time. September 17, 1958 to May 27, 1960.
Tuition $2,020.00 (two years). Instruments and books approximately $231.00.

No. 122 C. Two academic years, half time. September 17, 1958 to May 27, 1960. (Two and one half days weekly.)
Tuition $1,010.00 (two years). Instruments and books approximately $135.00.

No. 122 D. Four academic years, half time. September 17, 1958, to May 25, 1962. (Two and one half days weekly.)
Tuition $2,020.00 (four years). Instruments and books approximately $231.00.

These courses are designed for comprehensive preparation in Periodontia and Oral Medicine and are of particular significance for certification towards specialization. All didactic and clinical phases are covered with the objective of developing expert proficiency.

No. 222. GRADUATE COURSE IN PERIODONTIA AND ORAL MEDICINE.

Two academic years, full or half time, beginning Sept. 17, 1958. Tuition $2,020.00. (two years). Instruments and books approximately $275.00.

This program leads to the degree of Master of Science (in Dentistry). The objectives are to teach superior proficiency in Periodontia and Oral Medicine based on adequate background in the basic sciences.

For further information, write to the Secretary, Postgraduate Studies, New York University College of Dentistry, 421 First Ave., New York, N. Y.

UNIVERSITY OF MINNESOTA SCHOOL OF DENTISTRY

The 2nd Annual Course in Advanced Oral Pathology will be given by the University of Minnesota during the week of April 20-24, 1959. The Course consists of both lectures and laboratory. The subjects and faculty will include: Tumors of the Skin—Dr. Herbert Z. Lund, Professor of Dermatology, University of North Carolina; Tumors of Soft Tissues—Dr. Raffaele Lattes, Professor of Surgical Pathology,

Columbia University; Skin Dermatoses—Dr. Robert Goltz, Assoc. Professor of Dermatology, University of Minnesota; Non-Neoplastic Diseases of the Jaws —Dr. William Shafer, Professor of Oral Pathology, Indiana University; Tumors of Bone—Dr. David C. Dahlin, Assoc. Professor of Pathology, Mayo Clinic; Radiation Pathology—Dr. Donn Mosser, Professor of Radiology, University of Minnesota; Advanced Roentgenographic Diagnosis—Dr. Edward Stafne, Professor of Dentistry, Mayo Clinic; Salivary Gland Pathology—Dr. Anand Chaudhry, Associate Professor of Oral Pathology, University of Minnesota; Odontogenic Lesions—Dr. Robert Gorlin, Professor of Oral Pathology, University of Minnesota.

The tuition will be $65.00. The Course registration is limited to 15 individuals. Housing for the five days will be approximately $15.00. Interested individuals should immediately contact:

Dr. Robert J. Gorlin, Chairman, Division of Oral Pathology, School of Dentistry, 242 Owre Hall, University of Minnesota, Minneapolis, 14, Minnesota.

MICHAEL REESE HOSPITAL

Dr. Emanuel Cheraskin, Professor and Head of Department on Oral Medicine, University of Alabama will present a two day course on the physiology and pathology of blood and urine, tests for infectious diseases, chemical intoxication, neoplasia, stress syndrome, nutrition and hormonal deficiencies, and illustrate how they may be utilized in the practice of dentistry. Dr. Arthur Elfenbaum, Professor Emeritus, University of Illinois and Northwestern University and in charge of Diagnostic division, Department of Dentistry, Michael Reese Hospital will correlate Dr. Cheraskin's presentations to diagnosis and x-ray interpretation in the dental office.

The fee for the two day course will be $35.00, and will be given on Thursday and Friday, Feb. 12-13, 1959.

For further information and the detailed program write to Dental Department, Michael Reese Hospital 29th Street and Ellis Avenue, Chicago 16, Illinois.

THE WILLIAM J. GIES FOUNDATION FOR THE ADVANCEMENT OF DENTISTRY, INC.

Dr. Walter Hyde received the first William J. Gies Editorial Award for his editorial, Throw-Aways, which appeared in North-West Dentistry, July 1957.

Dr. George C. Paffenbarger, a member of the Board of Directors of the William J. Gies Foundation for the Advancement of Dentistry, presented a plaque to Dr. Hyde at the Annual Meeting of the American Association of Dental Editors in Dallas, Texas. In congratulating Dr. Hyde for adding to his numerous contributions to dentistry, Dr. Paffenbarger pointed out that one of the purposes for which the Gies Foundation was founded, and among William J. Gies' primary interests, was the promotion and aid of non-proprietary dental journalism.

This award, which includes a prize of two hundred dollars, will be an annual event. Graduate dentists are invited to submit editorials published in non-proprietary dental journals in the United States and Canada for the 1958 award. Papers should be limited to 750 words, appear in the 1958 volumes of the journals, and submitted to the Chairman of the Editorial Award Committee, Dr. John P. Traugott, 121 East 60 Street, New York 22, New York, by February 1, 1959.

NEW YORK UNIVERSITY COLLEGE OF DENTISTRY

New York University's College of Dentistry, through its department of endodontia, will offer a 40-hour postgraduate course during March, 1959 on new developments in the treatment of vital and non-vital teeth.

Participants in the course may attend morning and afternoon sessions for one week or morning sessions only for two weeks. The lectures and demonstrations begin on Monday, March 9.

Through clinical practice on patients, the course deals with the methods of treating pulp-involved teeth to maintain them in a healthy, functioning condition. It also includes a review of new developments in endodontics and the related sciences of pathology, Bacteriology, pharmacology, radiology, and restorative dentistry.

All classes will meet in the College's recently-acquired, ten-story building at 421 First Avenue in Manhattan. Instructing will be Dr. Morris Auerbach, chairman of the department of endodontia, and members of the department's postgraduate staff.

Further information can be obtained by writing to the Postgraduate Division, New York University College of Dentistry, 421 First Avenue, New York 10, New York.

TUFTS UNIVERSITY SCHOOL OF DENTAL MEDICINE

Tufts University School of Dental Medicine announces the following Postgraduate Refresher Courses.

DPG. 302—LOCAL ANESTHESIA
Monday, Tuesday, Wednesday
February 9, 10, 11, 1959
Tuition $75.00—Class limited to 15

Dr. Leonard M. Monheim, Professor of Anesthesiology, University of Pittsburgh, School of Dental Medicine

DPG. 602—OCCLUSAL EQUILIBRATION
Monday, Tuesday, Wednesday
February 9, 10, 11, 1959
Tuition $100.00—Class limited to 12
Dr. Edward T. Fischer, Assistant Professor of Periodontology

DPG. 301—MINOR ORAL SURGERY
Each Wednesday for the Eight Weeks from
March 4 to April 22, 1959
Tuition $150.00 — Class limited to 15
Dr. Richard Taylor, Professor of Oral Surgery, Chairman of the Department
Dr. Max Jacobs, Associate Professor

DPG. 603—OCCLUSAL ADJUSTMENT
Its relation to dental, periodontal and temporomandibular joint problems
Monday, Tuesday, Wednesday
March 30, 31, April 1, 1959
Tuition $100.00—Class limited to 12
Dr. Sigurd Ramfjord, Associate Professor of Periodontics, University of Michigan School of Dentistry

DPG. 606—CLINICAL ADJUSTMENT OF THE OCCLUSION
Thursday, Friday and Saturday
(Saturday till 12 noon)
April 2, 3, 4, 1959
Tuition $100.00—Class limited
Dr. Bernard Jankelson, Diplomate, American Board of Prosthodontics; Clinical Associate and Lecturer University of Washington

DPG. 605—A SPECIAL PARTICIPATION COURSE IN PERIODONTOLOGY
Monday through Friday for One Week from
April 13 through April 17, 1959
Tuition $150.00—Class limited to 12
Dr. Irving Glickman and Staff

For further information and application write to: Director of Graduate and Postgraduate Studies, Tufts University School of Dental Medicine, 136 Harrison Avenue, Boston, Massachusetts.

NEW YORK UNIVERSITY COLLEGE OF DENTISTRY

The Postgraduate Division of the Prosthetic Department of the New York University College of Dentistry is privileged to announce a reception and dinner honoring Dr. Joseph S. Landa for his 33 years of leadership as a teacher, clinician and researcher in dentistry and for his humane, dedicated and benevolent service in the interests of his colleagues, his community and his country.

The reception and dinner to honor Dr. Landa will be held on Thursday evening, January 22, 1959 at the Hotel Belmont Plaza, Lexington Avenue at 50th Street in New York City. Subscription is $10 per person.

Please mail reservations to Dr. Sidney I. Silverman, *Chairman,* 80 Park Avenue, New York 16, N. Y.

UNIVERSITY OF ALABAMA SCHOOL OF DENTISTRY

The University of Alabama School of Dentistry will present three refresher courses during the month of February. Miss Gertrude Sinnett, B.S., D.H. will conduct a course in *"Dental Assisting"* on February 14 and 15; Irving R. Hardy, D.M.D. will present a program in *"A Practical Approach to the Full Denture Problem"* on February 21, 22, and 23; and Samuel D. Gore, D.D.S., will instruct a program in *"Removable Orthodontic Appliance Construction"* on February 28, March 1 and 2. (This course is limited to practicing orthodontists.)

A program in *"Occlusal Adjustment: Its relation to dental, periodontal and temporomandibular joint problems"* will be presented by Sigurd Ramfjord, L.D.S., M.S., Ph.D., on March 7, 8, and 9, 1959.

Two courses are planned for the month of April. On April 3, 4, and 5, *"Partial Denture Prosthesis"* will be conducted by William L. McCracken, D.D.S., M.S.; and on April 10, 11, and 12, *"Oral Medicine"* will be offered by Emanuel Cheraskin, D.M.D., M.D.

For further information, please contact the Director, Refresher Course Program, University of Alabama School of Dentistry, 1919 Seventh Avenue South, Birmingham, Alabama.

NORTHEASTERN SOCIETY OF PERIODONTISTS

Formation of the Northeastern Society of Periodontists was recently announced by Dr. Harold J. Leonard, Secretary of the American Board of Periodontology and first president of the newly formed group. The society is composed of approximately fifty members who specialize in Periodontology. Dentists within a radius of 400 miles will be invited to participate in an annual all day scientific session devoted to clinics, demonstrations and discussion of new techniques in Periodontology.

The officers of the society include:

President.....Harold J. Leonard
Vice-President.....Leonard S. Hirschfeld
Secretary.....Irwin W. Scopp
Treasurer.....George J. Whinston
Editor.....Norman E. Alderman

For further information, contact Dr. Harold J. Leonard, 745 Fifth Avenue, New York, N. Y.

ANTHONY POSTERARO, *Publicity Chairman*